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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

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THE RATIONALE AND RESULTS OF SIMPLE MASTECTOMY PLUS RADIOTHERAPY IN PRIMARY CANCER OF THE BREAST*

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"There never has been a period of more controversy concerning the treatment of breast cancer than at present. Indecision as to the most acceptable forms of treatment is expressed even by physicians with the widest experience. . . . Some believe that radical mastectomy . . . offers the highest chance for cure . . . others believe that simple mastectomy when followed by irradiation makes radical surgery no longer necessary." (Pack, 1940)

THE problem of cancer of the breast is fundamentally the problem of cancer outside the breast. When the disease is confined to the breast itself, simple mastectomy alone will cure; indeed, in strictly localized or biologically favorable cases, simple excision of the tumor will cure. When the disease has spread beyond the breast, radical mastectomy of the Halsted type will cure only in that group of cases in which the spread is still confined to the removable tissues of the chest wall and axilla. Data on long term survival rates, and recent observations on the frequency of internal mammary lymph node involvement, indicate that this "locally confined" group with extramammary involvement is relatively small, being about 10 per cent of all patients admitted with primary tumors.

In the prolonged dissection required in removal of the breast, pectoral muscles and axillary contents en bloc, it is believed by many surgeons that cancer emboli are sometimes spread via lymphatics to nonremovable lymph nodes, and via blood vessels to distant organs.[3,9,11] As Haagensen[15] observed, "All surgeons who have operated on far advanced cases have seen widespread generalized metastases develop soon afterwards, just as if a shower of carcinoma emboli had been set free by the operation. This of course is what a surgeon should expect if his dissection is carried into or through carcinoma." Does the number of instances in which this occurs counterbalance the number of cases saved by block dissection? *If* it does, then the radical operation penalizes as many as it saves, and the over-all salvage rate is not improved. In terms of absolute "cure" rate, simple mas-

* Delivered by invitation before the New York Cancer Society, at the Academy of Medicine, New York, New York, November 10, 1953.

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FIG. 1.—*Cancer of the breast, disseminated through the chest wall following radical mastectomy.* Patient, aged sixty-five (S. F. Hosp. No. 66242). Radical operation in April, 1951, for rapidly growing tumor of right breast (allegedly present only about six weeks). Tumor adherent to skin; nipple retracted; probable Stage III lesion. Radical operation performed despite these clinical signs of incurability. Microscopic evidence of muscle invasion; 7 of 18 lymph nodes studied showed cancer. No postoperative radiotherapy requested.

At time of re-admission with these chest wall nodules, a chest roentgenogram showed right pulmonary metastases. Consultative Tumor Board recommended palliative radiotherapy. However, ward physician wished to use hormone therapy. Given stilbestrol, with marked nausea; tried to maintain a dose of 5 milligrams t.i.d. without success. Died four months after admission, or one year after the radical operation, with generalized disease.

tectomy should be fully as effective. However, in terms of palliation and prevention of local chest wall recurrences simple mastectomy alone will be inferior; in order to match or surpass the results of radical operation it needs the addition of vigorous irradiation.

Before attempting to discuss these questions, it is agreed that the "spectrum" of breast cancer is so broad one would need many hundreds of personally observed cases, treated under control conditions, to warrant reliable conclusions. Like other cancers, breast lesions appear to fall into three general groups: (a) those that have already metastasized when first found; (b) those that have not yet metastasized, but will do so in the near future; and (c) those that will remain localized for years, without apparent tendency to spread. Macdonald[26] reported that some 56 per cent of cases with primary tumors *less than 1 cm. in diameter* had already spread to the axillary nodes; in about one-half of these cases with involved axillary nodes the internal mammary chain of lymph nodes must also have been involved.[17] At the other end of the spectrum, almost one-fourth of the cases with primary tumors over 5 cm. in diameter observed by Macdonald had apparently not spread. He concluded that "the biological potential of breast cancer is determined early in the pre-clinical phase of the disease and probably in the actual inductive phase of neoplasia. This inherent growth potential, balanced against whatever defensive reactions are excited in the host, constitutes a biological complex which determines the natural history of the tumor."

To illustrate the small size of the group in which the radical operation is regarded as decisive in terms of survival we may examine the series of *1,056 cases* of primary cancer of the female breast recently reported by Shimkin, Lucia, Stone and Bell[41] from the San Francisco area. The clinical staging (Portmann), and the five and ten year survivals in this group were as follows (Table 1).

For Stages I to III the survival rates were calculated from the date of operation (usually radical mastectomy), and for Stage IV from the date of admission. Considering the group as an even 1,000 cases, Table 1

Table I

Stage	Per Cent in Each Stage	5 yr. Survival (as per cent of entire group)	10 yr. Survival (as per cent of entire group)
I	32	23	19
II	28	12	7
III	30	5	2
IV	10	0	0
Total	100	40	28

Table II

POTENTIAL 5 YEAR BENEFITS OF RADICAL MASTECTOMY IN 100 CONSECUTIVE CASES OF PRIMARY BREAST CANCER

Stage	I and II	III	IV
No. in stages	55	30	15
Operable	Yes	?	No (distant metastases)

Axillary nodes negative in 40 per cent (22 cases): survival rate 80 per cent (18 cases).

Axillary nodes positive in 60 per cent (33 cases): survival rate 30 per cent (10 cases).

Conclusion: Radical procedure useful in at least 10 cases.

shows that some 400 women survived five years, and 280 ten years. Note that 230 of the five year survivors were Stage I, that is, had tumors microscopically confined to the breast; most of these would presumably have been salvaged equally well by simple mastectomy. Some 120 of the five year survivors were Stage II, with microscopic evidence of tumor in the regional axillary lymph nodes; these women may be considered as salvaged by the radical operation, but note that they potentially dwindle to *70* at ten years; that is, the tumors in 50 cases were not eradicated. Next, about 50 of the Stage III cases survived five years; these potentially dwindle to *20* owing to recurrence after the five year period.

In other words, there were 70 plus 20, or 9 per cent of the entire group of cases, apparently salvaged for ten years, thanks to removal of extramammary carcinomatous tissue. At fifteen and twenty years, this proportion will doubtless dwindle further (as shown by Lewis and Rienhoff[23] in a study of Halsted's own material). Nevertheless it does represent a worthwhile achievement.

In terms of five year salvage, the data of Shimkin *et al.* indicate that 12 per cent of the entire group of cases were in the Stage II survival group, apparently arrested as a result of the radical operation. This is close to Smither's[45] estimate of 100 cases as may be shown by Table II.

The impression that radical operation may increase the chance of metastasis is supported by studies on a random sample of 100 out of 450 cases dead of breast cancer recently reported from the University of Pennsylvania.[19] The appearance of metastasis or recurrence was earliest in the radically operated group; it *seems* to have been delayed by postoperative irradiation in the group so treated (Table III).

Table III

Treatment Procedure	Mean Number of Years between Estimated Onset of Disease and Appearance of Recurrence and/or Metastasis
None	0–1 yr.
Roentgen irradiation	2–3 yr.
Surgery alone	1–2 yr.
Surgery plus roentgen irradiation	2–3 yr.

DEFINITIONS

To reduce misunderstanding, it may be desirable for purposes of this discussion to define our terms.

Radical operation is held to mean the removal, en bloc, of the skin (at least 8 cm. from the palpable edges of the tumor), the tumor, the pectoralis major and minor muscles, the axillary fat and lymph nodes, and the tributaries of the axillary vein. It does not include the superradical procedures of supraclavicular or anterior mediastinal excision.

Simple operation means the removal of the tumor and breast via a limited skin in-

cision, usually transverse. McWhirter[30] advises that the pectoral fascia be not removed, except when the tumor is firmly fixed; the muscle is then removed with the breast. Many surgeons prefer to remove the pectoral fascia routinely. Superficial, mobile subpectoral nodes are removed, but axillary dissection is not performed.

Postoperative radiotherapy, as usually employed, means the use of external roentgen therapy to at least two large fields over the axillary and supraclavicular areas, delivering a dose of 2,000–3,000 roentgens, in air, to each field, or a tissue dose of 3,500–4,500 roentgens in a period of three to four weeks. The treatment starts about ten days after operation.

Postoperative radiotherapy by McWhirter's method means that about twelve days following simple mastectomy, external roentgen therapy is given daily to four fields as follows: the patient's chest wall (and internal mammary and low axillary areas) are cross-fired by two long tangential fields, 10 by 15 cm. in size. The axilla and supraclavicular areas are cross-fired by two rectangular fields, about 12 by 25 cm. The patient is treated daily, except Sundays, for fifteen treatment days. Using 250 kv., a tissue dose of about 3,750 r is delivered to the potential tumor area. The half-value layer is 2 mm. copper for the chest wall and 3.7 mm. copper for the lymph node areas. (Note that neither of these techniques includes a really vigorous radiotherapeutic attack on the internal mammary lymph node areas.)

STAGING

The staging of disease we have used in our own practice for many years is based on that of Steinthal, modified by the findings at *roentgenographic* and at subsequent surgical examination. In other words, it is a clinical and microscopic method. It is not satisfactory, since it gives little clue as to the biological malignancy of a given tumor or the true degree of spread. A Stage I tumor is essentially a movable tumor that is confined to the breast; Stage II includes a movable tumor with palpable, mobile axillary nodes; Stage III comprises tumors with evidence of fixed nodes, or of spread beyond the axilla, or with extensive skin invasion; it includes Stage IV lesions of other classifications.

It is difficult to envisage any really satisfactory staging method in the present limited state of our knowledge. We know that about 30 per cent of cases of primary breast cancer without palpable axillary lymph nodes have microscopic evidence of lymph node involvement at operation. Conversely, about 30 per cent of cases with palpable nodes have no microscopic evidence of tumor in such.[15,41,42] Some 34 per cent of clinically operable cases have involved internal mammary lymph nodes.[17,27,48]

Further, of the cases diagnosed by qualified pathologists as axillary lymph node free after radical operation, it has been found in one series that about 30 per cent did actually have microscopic lymph node involvement on careful serial study.[40]

Since we referred earlier to Portmann's classification, it might be well to set down our understanding of it: Stage I represents cases with cancer localized to the breast, without involvement of the skin or lymph nodes, or attachment to deeper structures. Stage II includes similar cases, but with some axillary lymph node involvement. Stage III includes cases with skin involvement, satellite tumors, fixed tumors, and extensive axillary lymph node involvement. Stage IV consists of cases with evidence of distant metastases.

In view of the fact that biopsies of three sites are now recommended by some surgeons prior to staging certain cases,* and that prolonged and skillful histopathological study frequently increases the yield of positive lymph nodes, thereby leaving virtually no limit to microscopical possibilities, it may be well to re-appraise simple

* In patients with inner quadrant, or large centrally located tumors, or those with clinical evidence of axillary metastasis, Haagensen[27] performs under general anesthesia an incisional biopsy of the primary tumor, a biopsy of the first three intercostal space lymph nodes, and a supraclavicular lymph node biopsy.

clinical classification. Williams and Murley[51] recently reviewed the findings in 1,044 cases of primary female breast cancer seen at St. Bartholomew's Hospital and found to their surprise that in their material clinical staging was more valuable than pathological in assessing prognosis.[52] Of 164 cases, 29 per cent were in clinical Stage I but microscopic Stage II (axillary lymph nodes positive); 23 per cent of clinical Stage II had glands free of disease. However, the actual results of radical mastectomy paralleled the clinical findings. That is, clinical Stage II cases with negative lymph nodes had five year survival rates which were similar to those of verified Stage II cases. They conclude with the quite heretical statement: "Clinical assessment of the axilla may afford a more accurate clue to prognosis than might be expected."

If radical surgery is contemplated in a given case, we believe that McDonald, Haagensen and Stout's[27] recently modified criteria of inoperability must be noted. They believe that breast carcinoma is inoperable:

1. When extensive edema of the skin over the breast (more than one-third of the skin area) is present.
2. When satellite nodules are present in the skin over the breast.
3. When the carcinoma is of the inflammatory type.
4. When any *two, or more,* of the following signs of locally advanced carcinoma are present:
   a. Ulceration of the skin.
   b. Edema of the skin of limited extent (less than one-third of the skin over the breast).
   c. Fixation of the tumor to the chest wall.
   d. Axillary lymph nodes measuring 2.5 cm. or more, in transverse diameter.
   e. Fixation of axillary lymph nodes to the skin or the deep structures of the axilla.
5. When there is edema of the arm.
6. When, in patients with clinically involved axillary lymph nodes, a supraclavicular lymph node biopsy is positive.
7. When, in patients with clinically involved axillary lymph nodes, an internal mammary lymph node biopsy is positive.
8. When the patient has a history of recently developing pain in the back or pelvic region suggesting metastases, or roentgen examination reveals such.
9. When roentgenographic study of the lungs reveals metastases.
10. When palpation of the liver suggests that it contains metastases.

The clinical extent of most cases of primary breast cancer is obviously greater than suspected in a majority of cases submitted to radical operation. How and where has the tumor spread?

The spread of breast cancer is commonly thought of as essentially lymphatic and largely via the axilla; hence, the classical operation of Halsted.* However, in addition to these axillary lymph channels, it is now apparent (a) that vascular routes are all too common, even at the earliest stages, and (b) that other lymphatic pathways are often involved. Gil y Gil[12] reports that by routine sternal puncture he finds many metastases in cases clinically Stage I. Willis[52] reports the microscopic penetration of capillaries and small veins is frequent—a venous and capillary permeation comparable to that of small lymphatics. These vascular emboli give rise to silent metastases, notably in lungs, liver and bones. In 60 early postoperative cases recently studied by Goode and Martin[13] such blood-borne metastases outnumbered lymphatic metastases 3 to 1 (Table IV).

McKinnon[28] believes that "the blood spread of remote metastases occurs very early in the vast majority of cases and before the primary lesion is detectable." Gatch and Culbertson[11] note that venous dissemination of breast cancer is common and often early.

Batson[6] has demonstrated the existence

* It is now generally agreed[1,11,27,42] that breast cancer spreads by lymphatic and vascular emboli more often then by lymphatic permeation, as was once held by Handley and Halsted.

TABLE IV

PRESUMABLE ROUTE OF METASTASIS AFTER RADICAL MASTECTOMY*
(60 patients)

First Evidence of Persistent Breast Cancer via *Lymphatic* Spread		via *Hematogenous* Spread	
Skin of operative area	5	Bones	18
Chest wall, pleura, mediastinum	6	Lungs	12
Cervical lymph nodes	4	Distant soft parts	6
Other breast	2	Liver	5
		Brain	2
Total	17	Total	43

* This table was made using only those records that left little doubt as to the location of the earliest metastasis or first evidence of persistent cancer.

of a system of vertebral veins extending from the coccyx to the skull. These are valveless, thin walled and capacious, communicating freely with veins in the ribs, shoulder girdle, pelvis, and the upper ends of the humeri and femora. He was able to fill this system completely by injecting radiopaque material into a superficial vein of the breast. Bone metastases of breast cancers are in locations which emboli of cancer cells can easily reach by way of these veins without passing through the lungs.

Since the lymphatic drainage of the breast is complex and a subject of continued study, it may be well to note current surgical concepts of its nature. A primary breast cancer may spread by the following principal lymphatic pathways: the axillary, the pectoral and the internal mammary. The axillary route is the main route from the entire breast; its highest aggregation of lymph nodes is the *subclavicular group*. The pectoral (transpectoral and interpectoral) lymphatics also drain into this group. The subclavicular group then drains into the confluence of the internal jugular and subclavian veins either directly (via a "grand central lymphatic terminus") or indirectly (via a final sentinel inferior deep cervical lymph node) which overlies the venous confluence and empties therein. When other supraclavicular lymph nodes are involved by metastasis, the process is a retrograde one from the sentinel inferior deep cervical lymph node—and it is believed that the disease had already reached the grand central lymphatic terminus.

The internal mammary lymph nodes also drain into this terminus, and thence directly (or via the deep cervical lymph node) into the confluence of the internal jugular and subclavian veins. Cancer may then spread to the supraclavicular lymph nodes by *retrograde embolism*. That many cases with such internal mammary lymph nodes have bilateral involvement is suggested by the fact that in a majority of 13 patients dying with breast cancer, Ju *et al.*[20] found metastases in *both* chains of internal mammary lymph nodes.

Haagensen emphasizes that when the *subclavicular* or the *supraclavicular* group of lymph nodes are involved there is already nearly always some venous embolic spread, and operation will not cure. The same applies to most cases with internal mammary lymph node involvement.

Frequency of lymph node involvement in cases submitted to radical surgery. The most commonly recorded figure is 60 per cent for the *axillary area*. Even in cases with a history of a lump in the breast of less than one month's duration, Haagensen and Stout[14] found 50 per cent showing such metastasis; this increased to 68 per cent for those with breast tumors for more than six months.

For the internal *mammary area*, about 34 per cent of cases regarded as operable by current surgical standards are found to have involved lymph nodes.[17,48] The incidence is naturally higher with inner quadrant lesions. In axillary lymph node positive cases, Andreassen and Dahl-Iversen[3] found that 33 per cent had occult internal mammary lymph node involvement.

In summary, then, staging is difficult because of the fact that neither clinical, microscopic nor roentgenographic examination will permit the detection of the true degree of spread in many cases of primary breast cancer when first seen. Clinical stag-

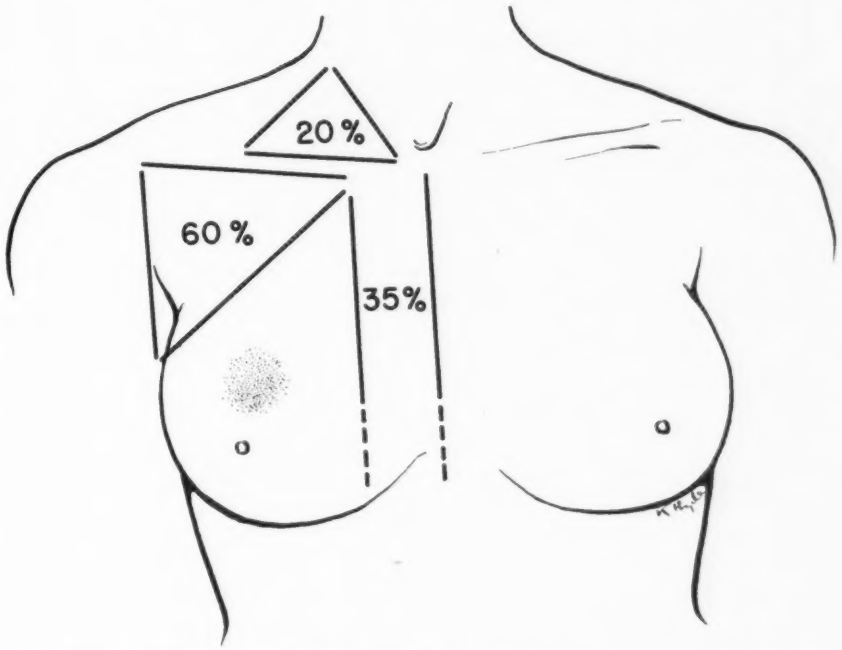

Fig. 2. Diagram showing the *incidence of involved metastatic nodes in "primary operable" cases of breast cancer.* The diagram is based on data published by Handley, Haagensen, Sugarbaker, Urban, and others. It refers to tumors of all quadrants considered as a group.

ing in experienced hands has apparently a moderately good correlation with surgical prognosis; microscopic staging in expert hands permits apparently valid decisions as to radical surgery in selected cases. None permits satisfactory prognosis in the individual case.

Reasons for Simple Mastectomy and Roentgen Irradiation

It is apparent from the above descriptions of the spread of breast tumors that a large percentage of cases are already beyond the scope of removal when first seen.[8] Not only will *radical* operation fail to cure; there is evidence that it may shorten average survival time by from five to ten months in some inoperable cases.[14] It is followed by disabling edema of the arm in from 5 to 10 per cent of cases, and by weakness of the arm of variable degree in perhaps 10 per cent. In less than expert hands, it has a mortality rate of about 1 per cent. The absolute five year survival rate is about 34 per cent in most published reports (Table v).

Table V

Results Obtained with Radical Mastectomy

(Assuming *100 unselected cases*, of which 55 per cent are Stages I and II, 45 per cent in Stages III and IV. Cf. Stanford data, Siris and Dobson.[42] Also that 60 per cent of Stages I and II cases have positive axillary lymph nodes; and that 80 per cent of lymph node free, and 30 per cent of lymph node positive survive 5 years)

Stage	I	II	III	IV
No. in Stage	25	30	30	15
Absolute 5 year survival rates	65%	38%	20%	2%
Survivals	17	11	6	±

Total survivors 34+

Simple mastectomy should control all cases still confined to the breast, and in such cases supplementary radiotherapy is not needed. Unfortunately, as with the surgeon, the radiologist cannot distinguish the cases with microscopic involvement of

the axilla from those without. Therefore, when the surgical procedure is simple mastectomy, all should receive postoperative irradiation. In an unselected group of "operable" cases (*i.e.*, Stage I and II of Portmann's classification), it is known that about 60 per cent will have involved axillary lymph nodes. The other 40 per cent of this "operable" group will not have microscopic evidence of axillary lymph node metastasis by ordinary study, but, since they cannot be detected by clinical means, all must be treated in this program. They will not be harmed by the standard McWhirter type of postoperative course of radiotherapy; indeed, they will have fewer complications than if radical dissection of the axilla had been performed. The absolute five year survival rate obtained by the chief proponent of this program is 42 per cent (Table VI).

TABLE VI

RESULTS OBTAINED WITH SIMPLE MASTECTOMY AND ROENTGEN IRRADIATION

(Assuming distribution of cases same as in Table V for radical operation)

Stage	I	II	III	IV
No. in Stage	25	30	30	15
Absolute 5 yr. survival rates	58%		29%	4%
Survivals	32		9	1

Total survivors 42

The fact that cancer cells are spread at the time of operation is now appreciated by many surgeons.[7,11] McDonald, Haagensen and Stout[27] recently re-emphasized the risk of transplanting such cells at the time of biopsy and operation. Goode and Martin[13] observe, "Although the value of surgical treatment for breast cancer appears obvious from the preceding observations, it is our opinion that in certain instances operative manipulation or incomplete removal of a tumor causes it to grow much more rapidly than it had been growing previously. The damage may be caused by squeezing viable cancer cells into intact lymphatics or blood vessels at the time of operation, or by the growth of incompletely removed cancer into the newly formed, delicate vessels found in the granulation tissue of the healing wound." We realize that this may occur with the simple operation, but it is obviously much more likely to occur with the radical procedure.[48]

On the other hand, Adair[2] does not seem to take much stock in the hazard. He writes, "we would prefer to perform 10 radical mastectomies in unfavorable cases than neglect to do the radical in the one in which we might be mistaken as to prognosis."

Many surgeons believe that simple mastectomy is unwise because "you don't know what's in the axilla." Well, first of all, knowledge of the axillary contents is only part of the whole problem (there are two other commonly involved but hitherto seldom tackled lymph node groups in the mediastinum and supraclavicular fossa). Second, even if one did "know," the results show that only about 30 to 40 per cent of axilla positive cases survive surgery for five years. These might have survived equally well with radical radiotherapy; some of them were undoubtedly of the biologically favorable type that would have survived for that length of time without any treatment at all[47]—dormant perhaps, as the internal mammary lymph nodes must be in many patients. If the program of simple mastectomy and postoperative radiotherapy is applied to all eligible cases, the clinical or microscopic *status of the axillary lymph nodes is of no importance in planning or executing treatment.* Further, if the results of the treatment program are calculated on an *absolute* basis, and deal with an adequate number of cases they may reasonably be compared with other *absolute* results.

It is reported by several observers* that,

*TABLE VII

Per cent of radically operated cases with internal mammary lymph node metastases

Author	*Inner Half and Central Lesions*	*Outer Half Lesions*
Handley	60	18
Haagensen	41	21
Urban	48	20

of patients hitherto submitted to radical operation, about 56 per cent have internal mammary lymph node involvement when the primary tumor is in the inner half of the breast, and about 20 per cent have such involvement when the primary tumor is in the outer half. These lymph nodes have been left intact by most surgeons to date. Yet some 50 per cent of women with Stage I and II tumors in the *inner* half of the breast live five years after radical mastectomy, and a slightly higher percentage with *outer* half tumors so survive. Many of them presumably harbor positive mediastinal lymph nodes (Table VIII).

TABLE VIII

SITE OF PRIMARY TUMOR AND RELATIVE FIVE YEAR* ARREST RATES BY RADICAL MASTECTOMY

(Stages I and II)

		Inner Quadrants	Outer Quadrants
Nohrman	(1949)	48%	52%
Haagensen	(1951)	49%	52%
Shimkin *et al.*	(1952)	62%	63%

* In several series of operated cases (Haagensen, Shimkin, etc.) about 36 per cent of primary breast cancers were located in the inner quadrants, including the central breast zone; about 64 per cent were located in the outer quadrants.

That there is a tendency to dormancy of metastatic carcinoma in some lymph nodes has been noted by Heyman in the lateral pelvic lymph nodes in clinically arrested carcinoma of the cervix. McDonald *et al.*[27] make this observation on the internal mammary nodes: "Nodes containing metastases were usually at least 4 or 5 mm. in diameter. It is noteworthy, however, that even at the end stage of disseminated carcinoma, with massive metastases in the viscera elsewhere, the involved internal mammary lymph nodes remained only from 1 to 2 cm. in diameter. The disease had presumably been present in the internal mammary region for a long time, yet it had not grown to sufficiently massive proportions to give any clinical or radiographic evidence of its presence in this region."

The Effect of Radiotherapy. The biological effect of irradiation is a resultant of many forces. The physical ones include the tissue dose in roentgens, the over-all time of treatment, and the volume of tissue irradiated. If breast cancer resembles epidermoid skin cancer in sensitivity, Strandqvist's curve may give a clue as to the influence of dose and time. He found that approximate equivalent cancerocidal roentgen-ray doses to small fields were: 2,500 r in one day, 5,000 r in fifteen days, and 6,000 r in thirty days. His data of course *cannot* be transposed to large fields or large volumes. From a fairly extensive experience with bulky skin cancers about the face, I know that a few are curable with doses as low as 3,500 r, tissue dose, in three weeks; some are curable with 5,000 r in four weeks. It seems to me that a similar state of affairs exists with breast tumors. There is no standard cancerocidal or cancer suppressive dose. It is not possible to deliver 6,000 r with safety to the entire breast and axilla in thirty days. It is possible to deliver a tumor dose of 3,500 to 4,500 r in two and one-half to five weeks to these areas, especially as a postoperative measure. This dose will arrest the radiosensitive tumors, and will usually depress or retard the more resistant ones. Very localized breast tumors may of course be treated more intensively; widespread chest wall involvement requires more moderate dosage.

There seems little question among surgeons that radiotherapy is of great benefit in Stage III breast cancer. As McDonald, Haagensen and Stout[27] have recently observed, "modern intensive protracted irradiation has great palliative value in the disease." Orr[35] notes that there is "abundant recorded evidence that irradiation will devitalize or destroy breast cancer cells." Halley and Melnick,[16] Richards,[38] and Lumb[25] have clearly demonstrated in a series of experimentally studied cases that radiotherapy can, in some instances, destroy all traces of carcinoma in a given breast tumor as studied by serial sections. The percentage so affected ranged from 16 per cent to 50 per cent in different series. In addition, Adair thought that axillary

lymph node metastases were destroyed in 22 per cent of one series he studied. In our own clinical practice we have seen many cases with long-term palliation and comfortable survival following radiotherapy.[10]

On the other hand, several surgeons are dubious as to its value in Stage II cases. This paradox is shown by Haagensen and Stout's[14] report on 38 "operable" cases treated by Lenz with tumor doses estimated as 4,500 r in eight weeks. At operation three months later, Stout found marked radiation effects but some cancer cells present in all cases.* Yet Lenz[22] himself reports 46 "inoperable" cases treated by roentgen therapy alone, with 10 of them clinically well after five years (22 per cent). Baclesse[5] obtained an even better survival rate by radiotherapy in Stage II and III lesions. As Rutihauser and Majno[39] have observed, there is a wide variation between the morphologic changes produced by irradiation and the clinical effects thereof. Viability of cancer cells can be proved only on biological grounds.

Smithers and his associates[45] sum up an extensive experience by noting that radiotherapy can "eradicate the disease in some cases and restrain growth, sometimes for long periods, in others." Watson has reported five year results in cases treated with and without radiotherapy and by radiotherapy alone. He writes: "At the present time it would seem that radiation therapy in Stage I cases gives about 5% less five year survivals than radical mastectomy. When the nodes are involved (Stage II) there does not seem to be much difference." (Personal communication, October 17, 1953.) Smith and Smith,[44] after reviewing 739 cases treated in Boston, concluded that their Stage II cases showed a statistically significant improvement in survival rates with postoperative irradiation (44 per cent five year survivals as compared to 22 per cent); they thought the improvement occurred chiefly in the Grade 3 tumors.

* Stout now agrees that the microscopic presence of tumor cells after adequate radiotherapy is *not* synonymous with viability of these cells.

It has been my privilege to observe the effects of radiotherapy in some 544 cases of primary breast cancer personally seen during the last twenty years; the radiotherapy was usually combined with radical surgery. One hundred and thirty-six of these cases were seen in clinic practice (Stanford Service, San Francisco Hospital), and 408 in private practice (St. Joseph's Hospital, San Francisco Hospital and the writer's office). The radiotherapy was largely external roentgen irradiation, using 200 kv. with a half-value layer of 1 mm. copper; in some cases, beams of softer quality were used for superficial lesions, and for a few years beams of harder quality were used for certain lymph node areas.

In 161 cases a short, wide field *preoperative* course of irradiation was given to the breast, chest wall and axilla (1,000 r, tissue, in four days); Dr. Leo Eloesser, the operating surgeon (then Clinical Professor of Surgery at Stanford University Medical School), believed that his incidence of local recurrence was greatly diminished by this treatment. However, statistical proof of such benefit was lacking, and the program was discontinued some years ago.

In 230 cases a moderately intensive *postoperative* course of irradiation was given to the axillary and supraclavicular area (up to 3,500 r, tissue, in three to four weeks, using opposing fields, 12.5 by 25 cm.). This was given largely to Stage II and III cases as a "prophylactic" or suppressive measure. In a small number of cases, the internal mammary lymph node area was also treated. We believe that this course helps those patients with radiosensitive tumors.†

In 146 cases the patient was treated by irradiation *alone* (with doses up to about 4,500 r, tissue, in four to six weeks, including the breast and regional lymph node areas). This was chiefly for advanced or inoperable lesions, for persons in whom

† With 200 kv., half-value layer 1 mm. copper, target-skin distance of 50 to 70 cm., an air dose of 2,000 r to each of two opposing supraclavicular axillary fields measuring 20 by 12 cm. gives a lymph node dose of about 3,500 r in 3 weeks in the average patient. With an air dose of 3,000 r to a single anterior mediastinal field, 18 by 7 cm., the internal mammary lymph node chain at 2 cm. depth receives about 3,500 r in that time.

operation was deemed inadvisable, or for persons who refused surgery (15 patients).

In 7 cases with Stage II lesions the patient was treated *during operation*, the exposed axilla being irradiated with a single dose of about 2,000 r before closing the incision. This was done as part of a clinical investigation with one surgeon. It was not successful and, in our opinion, should not be repeated.

In addition to the above primary cases of breast cancer, we had, of course, a large number of patients with recurrent postoperative and metastatic lesions to treat; not all of these were indexed or registered. Data are available on 95 locally recurrent postoperative cases, and some 260 cases with distant metastases (largely to bone, lung, pleura and brain). Our results in treating certain cases in this group have been published.

The reasons for simple mastectomy and radiotherapy may therefore be summarized:

1. Simple mastectomy is adequate for most lymph node free cases of breast cancer.

2. The procedure is safer and has a lower morbidity rate than radical surgery: there is less mutilation and deformity, and there is quicker wound healing.

3. Complications are fewer (for example, postoperative arm edema is usually absent, while it occurs in about 10 per cent of cases treated radically).

4. There is decreased chance of cancer

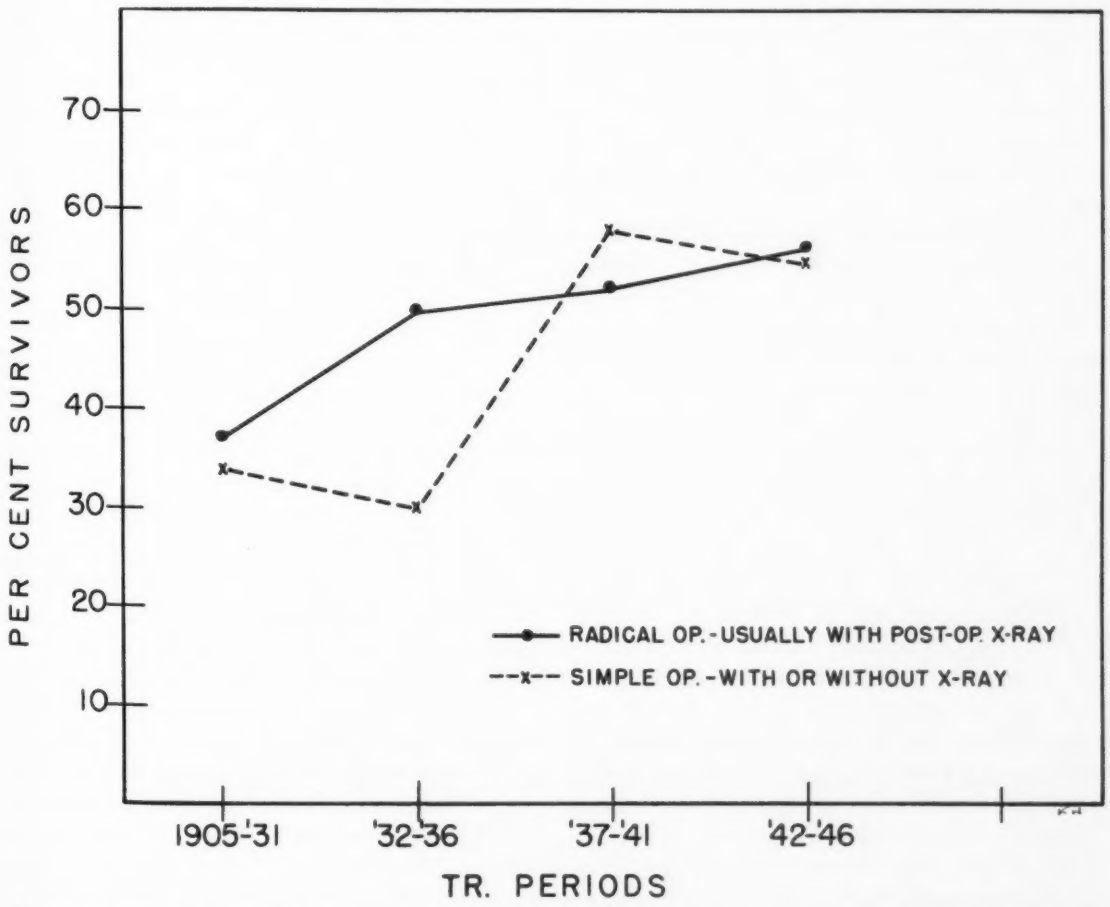

FIG. 3. The relative five year survival rates of patients subjected to *radical* or to *simple* operation, according to different periods, based on survival data in 739 cases reported by Smith and Smith, 1953. Note that just as the results of radical operation show steady improvement between 1930 and 1946, the results of simple operation do likewise, in the particular samples reported.

dissemination, since the prolonged handling of tissues necessary in block dissection of the axilla is avoided.

5. The patient may resume her occupation or useful work earlier than after the radical procedure.

6. The radiation therapy (being postoperative) does not delay removal of a potential "solitary focus" in the breast.

7. The five year absolute survival rate is as good as that generally reported with radical surgery.

The *disadvantages* of simple mastectomy and radiotherapy include the following:

1. Failure to control radioresistant, but locally removable extramammary cancer (for example, in the chest wall or axillary lymph nodes).[3,47]

2. Probable failure to secure adequate dosage in obese patients (McWhirter advises radical operation for such patients).

3. Possible hazard to the lung apex in patients with active upper lobe tuberculosis; radical operation will be preferable in such patients.

4. Possible radiation injury to the tissues when the procedure is not meticulously conducted.

5. Protraction of a treatment method for some two and one-half weeks in addition to the surgical time involved.

It may be argued that the results of radical mastectomy in the last five or ten years are much superior to those formerly attained, and therefore the place for simple mastectomy is increasingly small. Analysis of recent data from the Boston area[44] shows that the favorable upward trend in radical mastectomy survivals is actually paralleled by a similar trend in simple mastectomy results. The five year survival rates of *radical* mastectomy increased from 36 per cent in the period prior to 1932, to 56 per cent in the period subsequent to 1942. Conversely, the five year survivals of the *simple* operation increased from 34 per cent in the first period to 55 per cent in the last. Both sets of cases had postoperative therapy, as a rule. These figures refer to cases handled in the same hospital, but are, of course, not strictly comparable in number or quality. Nevertheless, they indicate a rising trend in short term survivals from the simple operation (Table IX).

THE RESULTS OF SIMPLE MASTECTOMY AND RADIOTHERAPY

The writer has had only a small personal experience with simple mastectomy and postoperative radiotherapy by McWhirter's method, all of it within the past five years. However, there is now a fairly large number of cases reported in the literature; these will be used as a possible indicator of results obtainable. Short of a large volume of material, completely followed, it is impossible to credit any particular treatment method with specific results.

TABLE IX

IMPROVEMENT IN RELATIVE SURVIVAL RATES (PER CENT)

(739 cases. Smith and Smith)

Period	(5) Years	(10) Years	(15) Years	(20) Years
Radical Operation				
1905–1931	36	25	14	9
1932–1936	48	30	18	
1937–1941	52	33		
1942–1946	56			
Simple Operation				
1905–1931	34	17	8	7
1932–1936	30	12	6	
1937–1941	58	32		
1942–1946	55			

Note: Most cases had supplementary radiotherapy.

Personal Material. Thirteen cases with biopsy proved primary carcinoma of the breast were treated in consultation with Colonel E. A. Lodmell and General L. D. Heaton at Letterman Hospital. The ages of the patients ranged from thirty-five to seventy years. There were 8 cases in Stage I, 4 in Stage II, and 1 in Stage III. All were treated by simple mastectomy followed by postoperative radiotherapy, using McWhir-

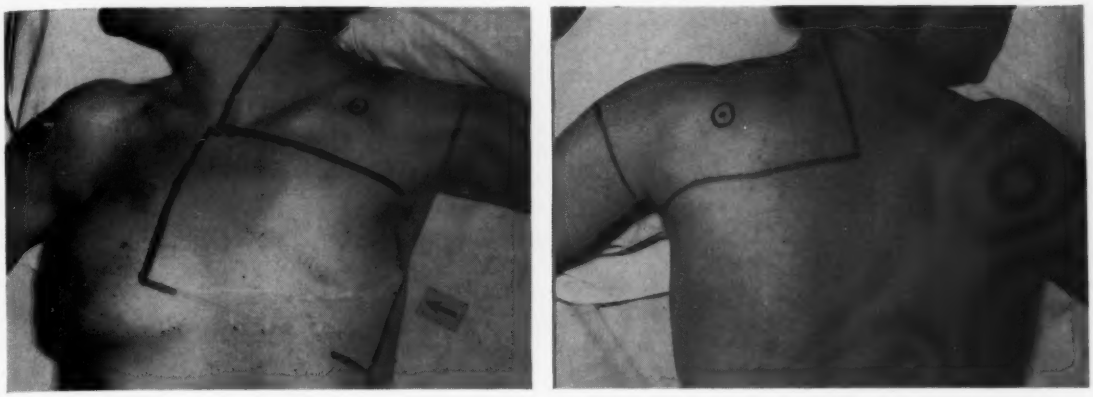

Fig. 4. Showing patient set-up for radiotherapy according to the McWhirter technique. The bolus bags are not shown. The anterior and posterior fields over the supraclavicular and axillary areas measure about 12 by 25 cm. The two tangential fields are 15 cm. in length and are irradiated through a 10 by 15 cm. beam. The total skin dose to each field is about 4,000 r in 15 treatment days. The estimated minimum tumor dose throughout the treated tissue is about 3,750 r; the maximum, about 4,500 r.

ter's technique. The following are the results to date (Table x).

The ages of the Stage I cases ranged from thirty-eight to seventy years. The 3 living Stage II cases were fifty-five to sixty-four years old; the 1 dead with disease was thirty-five years old. The 1 Stage III case

Table X

Stage	*Cases*	
I	8	All living and well (6 months to $3\frac{1}{2}$ years)
II	3	Living and well ($1\frac{1}{2}$ to $3\frac{1}{4}$ years)
II	1	Dead of disease (in 5 months)
III	1	Dead of widespread disease (in 1 month)

was aged sixty. Immediate reactions: these included erythema, with desquamation, in all cases.[24] Late complications: there have been none to date, except in 1 patient who was rather uncooperative, and failed to complete the last three treatments. She had mild infection of the operative site and now has slight edema of the arm. She has no evidence of recurrent tumor.

To these may be added a group of 14 patients we treated at two other hospitals by simple mastectomy plus radical postoperative radiotherapy; 5 of these are living and well over five years. The entire group may be summarized as follows (Table xi).

Table XI

Hospital	Number of Cases	Number in Stage			Number Living and Well
		I	II	III and IV	
Letterman	13	8	4	1	11 (2–4 yr.)
San Francisco	9	2	1	6	3 (6–8 yr.)
St. Joseph's	5	0	5	0	2 (5–7 yr.)
Total	27				16

Results in the Literature. In Table xii the results obtained by seven different radiotherapists are published; this table lists Stage I and II cases in order to be comparable with current radical surgical results in such cases. The results show a five year survival rate of from 58 to 80 per cent, the majority being in the 60 per cent range. The only significant number of cases is that reported by McWhirter, concerning which much has been written.[30,31,45]

The material of Williams and Murley[51] includes some radium treated cases. That of Mustakallio[33] pertains largely to simple surgery (local excision) and radiotherapy. The material of Shimkin *et al.*,[41] of Deaton and Bradshaw,[9] and of Orr[35] is of interest because it comes from clinics wherein cases

TABLE XII

RESULTS OF SIMPLE MASTECTOMY AND POSTOPERATIVE RADIOTHERAPY, STAGES I AND II

Cases	5 Yr. Survival	Author
	per cent	
165	62	Williams and Murley[51]
127	80	Mustakallio[33]
28	64	Shimkin *et al.*[41]
30	63	Deaton and Bradshaw[9]
15	60	Orr[35]
907	58	McWhirter[31]
66	64	Nelson, Perth Gen. Hosp., 1953

TABLE XIII

ABSOLUTE 5 YEAR SURVIVAL RATES (OPERABLE AND INOPERABLE CASES)

(McWhirter, 1952)

Stage	Simple Mastectomy and Irradiation	Radical Mastectomy and Irradiation
I and II	58%	48%
III	29	18
IV	4	2
Total	42%	31%
Cases	1,606	784

were treated mainly by radical surgery—with no better results. The respective five year survival rates in these three clinics using radical surgery, was 60 per cent, 55 per cent and 45 per cent. With simple operation plus radiotherapy they were 64 per cent, 63 per cent and 60 per cent. The results of the simple procedure plus radiotherapy are comparable to those published by Haagensen and Stout[14] and Harrington[17] for the radical operation; that is, about 61 per cent five year survivors in Stages I and II cases combined.

While these data are of interest, it is realized that it is not possible scientifically to compare the results obtained in two series of cases treated by different methods, except when the series are biologically similar and cared for concurrently—an almost impossible attainment in treating human cancer. Therefore, the results are suggestive and not conclusive.

Ten and twenty year data would be desirable. There are a moderate number of ten year data from McWhirter's clinic; his absolute results then are as good as those obtained by the radical procedure (25 per cent). Doubtless, the over-all results of simple mastectomy and postoperative radiotherapy will show the same steady decline reported in all long-term studies of treated breast cancer.[23,28,34,37,45]

Comment. The important problems facing any proponent of simple mastectomy are:

1. Will he be able to duplicate McWhirter's excellent results?

2. Will local chest wall and axillary recurrence rates *after* five or ten years be less than those following radical operation (which are about 12 per cent in well selected cases, and about 40 per cent in poorly selected ones)?[15]

3. Will late radiation changes (from improperly given therapy) discredit the method?

Slaughter and Peterson[43] observed that, "Most radical mastectomies are palliative in the sense that less than half of all breast cancers so treated are cured, although the operation originally was advised as a curative attempt!" I suspect that a similar statement must be applied in general to the simple operation combined with postoperative radiotherapy. Even Halsted felt that "the efficiency of the operation is measured truer in terms of local recurrence than of ultimate cure."

Preoperative Radiotherapy. Nohrman,[34] Ash *et al.*,[4] McNealy[29] and Goode and Martin[13] present persuasive arguments and data in support of preoperative radiotherapy in selected cases of breast cancer, especially those with low fixed axillary nodes. Space and time do not permit consideration of this phase of the problem in this paper.

Similarity of End Results, Irrespective of Treatment. Many writers have commented on the apparently small influence of treat-

ment on survival rates.[7,11,28,37] From St. Bartholomew's Hospital comes an interesting review of some 1,044 cases of primary female breast cancer treated up to 1940 by one of three general methods: simple surgery, modified radical surgery and radical surgery (all with or without radiotherapy). The results show that, in terms of survival, the various methods of treatment were about equally effective in Stages I and II. The authors comment that the evidence "must give rise to sobering thoughts about the value of extensive surgery. The type and extent of the disease at the time of treatment rather than the method of treatment rather than the method of treatment used is clearly the important factor affecting survival." In other words, "the good results are in the good cases."

Orr[35] reports on 322 cases treated at the University of Kansas. The five year survival rates for Stage I and II cases combined showed no significant difference with different types of treatment. The figures were: for simple operation 60 per cent; for radical operation 45 per cent; and for subradical operation 64 per cent.

Enthusiastic surgical groups, dealing essentially with Stage I and II lesions, give five year figures of 54 per cent (Adair), 52 per cent (Lahey) and 51 per cent (Harrington). Meanwhile, other students of the cancer problem, such as McKinnon, claim there is no decline in recorded breast cancer mortality in Massachusetts, Canada or Great Britain. He believes that "the spread of remote metastases occurs very early in the course of the disease before the primary lesion is detectable by any means available today or in immediate prospect, and thus before interference with that spread is possible."

McKinnon concludes that "early treatment neither prevents nor materially postpones death from cancer in any large proportion of cases. It provides mental and physical relief in cases of lethal breast cancer and cure in cases of nonlethal breast cancer—the one indistinguishable morphologically from the other."

TABLE XIV

COMPARISON OF 5 YEAR RESULTS OF TREATMENT

Author Year and Cases	Per Cent of Cases in Stages I and II ("operable")	Relative Survival Rate (per cent)	*Absolute Survival Rate* (per cent)	Absolute Clinical "Cure" Rate (per cent)
Haagensen 1949— 668	78	50	*47*	39
McWhirter 1949— 480	65	43	*41*	32
Nohrman 1949—1,042	77	42	*40*	—
Adair 1949—3,836	61	37	*35*	—
Smithers 1952—1,093	57	35	*35*	28
Windeyer 1949—1,091	52	35	*33*	—
Siris and Dobson 1946— 319	60	39	*33*	27

(All but last line calculated by Smithers.)

Park and Lees[37] believe that radical mastectomy is not of proved benefit in the cure of breast cancer; it may increase survival time and the proportion of apparent cures. They regard the intrinsic malignancy or growth rate of the tumor as the decisive factor in survival, and would discard the five year arrest rate as a discredited yardstick, substituting therefor an "index of curability." At the present time they believe that the five year survival rate of breast cancer (treated or untreated) is two-tenths the number with axillary metastases at the time of diagnosis. The mean survival time of patients would be used as the index. Kraus[21] attempts to refute some of Park and Lees' contentions in a subsequent publication, and draws a different set of conclusions from the data which they used. While disagreeing with these authors on ineffectiveness of early treatment, he does

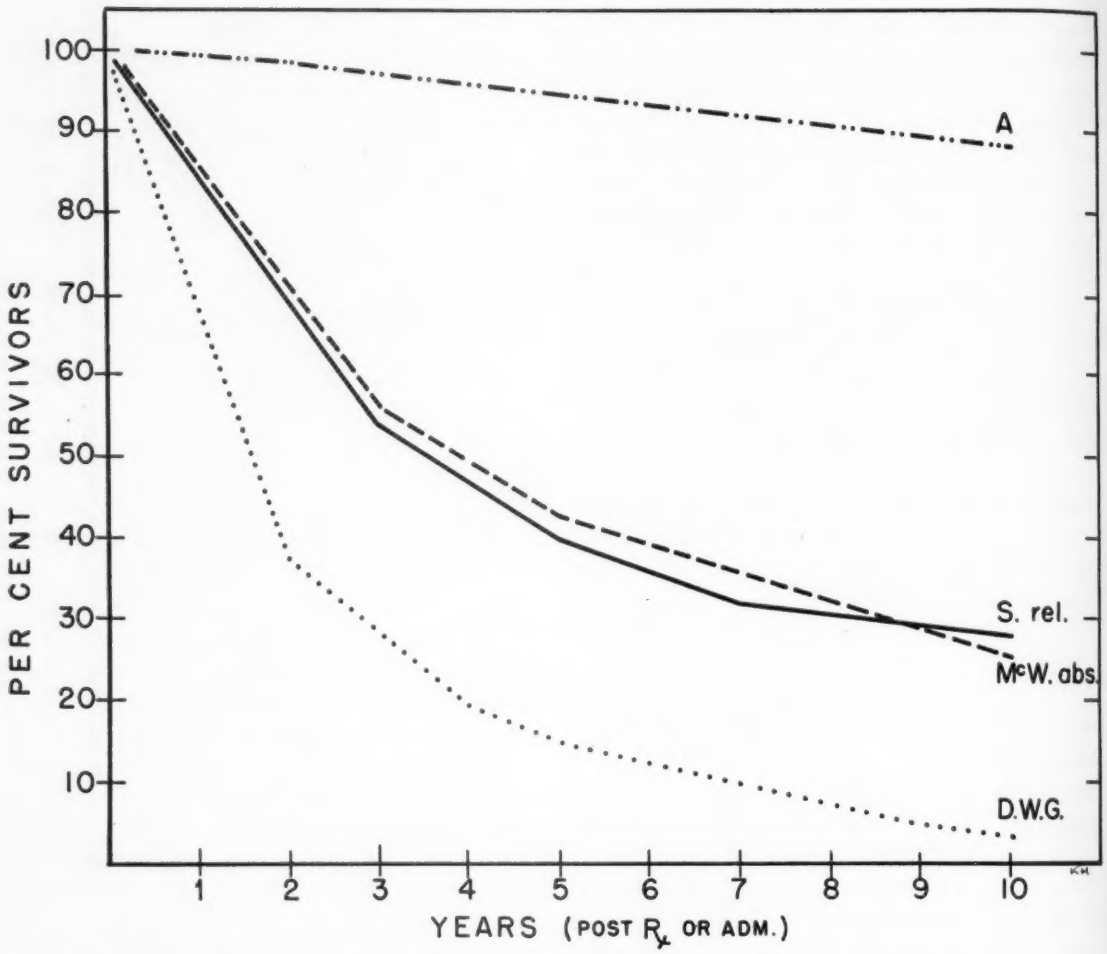

FIG. 5. Curves showing the ten year survival rates of: *A*. The general female population at age fifty-three during the decade ending 1945. *S*. The survival rate after radical mastectomy. (Relative survival rate of 899 women treated principally by radical mastectomy, and some with additional postoperative radiotherapy reported from the University of California in 1952.) *McW*. The absolute survival rate after simple mastectomy and postoperative radiotherapy. (Based on 1,606 women reported by McWhirter in 1952.) *D.W.G.* The apparent survival rate in untreated women with breast cancer. (Based on 616 cases reported by Daland, Wade and Greenwood, and corrected by the author to allow for the usual delay of about six months in treated cases reporting for actual treatment.)

The first curve refers to the general female population; the lower three curves to women with primary cancer of the breast. Note that the results of the two types of treatment closely parallel each other. The curves are intentionally weighted in favor of radical operation, since the radical results are relative, while the simple operative results are absolute.

concur with them on the great importance of variability of growth rate in different groups of cases. "The group showing higher survivorship contains a higher proportion of intrinsically lower degrees of malignancy." He does not controvert the apparent fact that, despite fifty years of radical surgery, the age-adjusted death rate for carcinoma of the breast per 100,000 persons in the State of Connecticut in 1937 was *30*, and in 1948, *37*. It is still rising.

It seems to me that a major weakness in Park and Lees' admirable review is their final conclusion: "If treatment is in any way effective, the effectiveness cannot be greater than that required to increase the over-all 5 year survival rate by more than 5 to 10 per cent." If it is true that the sur-

vival rate in untreated breast cancer is about 20 per cent, and that treatment adds 10 per cent to that figure, it is apparent that there is a real improvement, or one of about 50 per cent!

SUMMARY

In 100 consecutive women with primary cancer of the breast, some will be apparently operable when first seen, some questionably operable and some frankly inoperable. The proportions naturally vary in different localities, but in most reported series, about 60 per cent of cases fall into the first or apparently operable group, and about 40 per cent into the other two groups.

In the operable group, radical surgery results in some 80 per cent five year survivals when the axillary lymph nodes are negative for tumor, and in about 30 per cent such survivals when the lymph nodes are positive. Therefore, in this favorable group, there are some 30 women whose tumors are arrested for five years; of these, about 19 had negative lymph nodes (and would presumably have survived by simple operation alone), and about *11* had positive lymph nodes (and therefore represent an apparent salvage by the radical procedure).*

Radical surgery is contraindicated in most of the questionably operable and in all of the frankly inoperable cases. It may hasten the spread of fresh cancer emboli throughout the chest wall and body, and will actually shorten survival time in some patients.

There are therefore about 11 out of the 100 cases in which the radical operation is of significant value in terms of survival. This group will contain some patients with radiosensitive growths and some with radioresistant ones. It is our belief that adequate postoperative radiotherapy (about 4,000 r, tissue, in 15 to 21 days) will do more for the axillary lymph nodes in those members of this group with radiosensitive lesions than will radical dissection of the axilla. It will not spread neoplastic cells; it should destroy or suppress tumor. Conversely, radical dissection will do more for those members of the group with radioresistant lesions.

The question therefore is, what is the proportion of radiosensitive and radioresistant tumors in this hypothetical group of 11 patients? If it is evenly divided, either method should give equal end results in terms of over-all salvage. If radiosensitive lesions predominate, simple operation plus postoperative irradiation will have more to offer than radical surgery.

Since the radiosensitivity of lesions is not determinable prior to treatment, and since surgical removal is a more certain method of eradicating a localized (*i.e.*, removable) breast cancer than is radiotherapy, it is advisable that surgery be used to remove the primary lesion in all apparently operable cases; it permits identification of the tumor, serving as an excisional biopsy with minimum chance of disseminating disease.

The five and ten year survival rates in properly selected "operable" cases are essentially the same whether the treatment be radical mastectomy or simple mastectomy and postoperative radiotherapy.

The radical procedure is still preferable to the simple operation in a special group of patients with operable lesions (the *very obese* woman, the *apical tuberculoiic*, and the *unstable individual* who will probably fail to complete her postoperative therapy course). It is possible that light complexioned women under fifty years of age, with clinically favorable tumors (*i.e.*, of slow growth rate, apparently locally confined and therefore potentially well differentiated or radioresistant) also should have radical operation in preference to vigorous radiotherapy; possible late undesirable radiation changes in the skin will be obviated.

Patients with presumed internal mammary lymph node involvement (especially

* The "operable" group numbers 60; in most such groups 40 per cent are found to be without microscopically involved axillary lymph nodes (24 cases) and 60 per cent with such lymph nodes (36 cases). If 80 per cent of 24 cases survive five years, it equals about 19 cases; if 30 per cent of 36 cases survive, it equals 11 cases. Should the operable group be as large as Haagensen's (78 per cent), the number of lymph node-positive cases surviving would be 14, but few other clinics report so high an operability rate.

those with central or inner quadrant tumors) probably should have additional radiotherapy to this area.

The availability of expert radical radiotherapy must be weighed against that of radical surgery in estimating the practicability of the proposed program. It is probable that most women, given the choice and the knowledge that the chances of arrest *are* equal, would prefer the simple procedure.

CONCLUSIONS

1. The place of simple mastectomy and vigorous postoperative radiotherapy in the treatment of primary operable cancer of the breast appears to be well established. Where skilled radiotherapeutic service is available, I believe it is the method of choice in most cases seen in practice today.

2. It still remains to be confirmed that late recurrences in the chest wall and axillary areas are no higher with this combined procedure than they are with the radical operation. If this does not eventuate, the procedure, naturally, will require re-evaluation.

3. The long term control of cancer of the breast, in terms of absolute ten year survival rates, is as good with the simple procedure plus adequate radiotherapy as it is with the radical procedure; there is some evidence to suggest that, under average conditions of practice, it is actually better.

4. The physical comfort of the average woman treated by simple operation and postoperative radiotherapy appears to be greater than after the radical procedure. However, either method still fails to cure a majority of women with breast cancer today.

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SOME OBSERVATIONS CONCERNED WITH CARCINOMA OF THE BREAST*

PART III

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DURING the years 1923 to 1943, there were 1,034 patients with carcinoma of the breast seen at the Hospital of the University of Pennsylvania.

Part I of this present study[1] included the gross statistics of survival for 1,029 of these patients when treated by the general methods of operation and/or with irradiation. Part II[2] included the results of radiation treatment combined with operation.

SCOPE OF THIS REPORT

The present report will be concerned with an analysis of irradiation for recurrences or metastases, the effect of castration, and the effect of the size of the original tumor on survivals. The data in the tables are grouped as follows:

I. *Local Recurrences and/or Metastases:*

1. Effect of treatment of local recurrences and/or metastases developing after original operation, operation plus irradiation, or irradiation only on five year survivals (detailed study), Table XXII.
2. Effect of treatment, data from Table XXII, plus a calculation of percentages to show the proportions of the whole series developing local recurrence with or without metastases (summary), Table XXIII.
3. Comparison of five year survival rates for patients treated or not treated for metastases (summary), Table XXIV.
4. Effect on the local lesion by various quantities or radiation (roentgen rays), Table XXV.
5. Effect on the local lesion by various quantities of radiation (radium), Table XXVI.
6. Number and survival of patients having irradiation for recurrences or metastases added to other forms of radiation therapy given prior thereto, Table XXVII.

II. *Influence of the Ovaries on Breast Carcinoma:*

1. Effect of pre- or postmenopause (Table XXVIII).
2. Effect of castration before diagnosis of carcinoma of the breast, Table XXIX.
3. Effect of therapeutic castration combined with other irradiation routines. (Richards' Stage of Disease Classification for Carcinoma of Breast), Table XXX.
4. Same as number 3. (Portmann's Clinical Classification for Carcinoma of Breast), Table XXXI.
5. Number of patients developing carcinoma during pregnancy or lactation, Table XXXII.
6. Comparison of five year survival between patients having or not having therapeutic castration. (Richards' Stage of Disease Classification for Carcinoma of Breast), Table XXXIII.
7. Same as number 6. (Portmann's Clinical Classification for Carcinoma of Breast), Table XXXIV.
8. Effect of irradiation castration on local lesions, Table XXXV.

III. *Size of Original Tumor of Breast:*

1. Correlation of size of breast tumors with type of therapy—operation, operation plus irradiation, or irradiation only, and five year survival, Table XXXVI.
3. Correlation of size of breast tumors with type of irradiation plus operation, and five year survival, Table XXXVII.
4. Correlation of size of breast tumors with duration of symptoms, and five year survival, Table XXXVIII.

STUDY OF RECURRENCES AND/OR METASTASES

Table XXII shows that in the series of 1,029 patients with carcinoma of the breast, 423 had local recurrences. Of these

* From the Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania, and the Penn Mutual Life Insurance Foundation for the Study of Neoplastic Diseases, and supported in part by the Institutional Grant of the American Cancer Society.

† Clinical Fellow of the American Cancer Society.

Table XXII

LOCAL RECURRENCES AND METASTASES. INFLUENCE OF TREATMENT ON NUMBER AND PERCENTAGE 5 YEAR SURVIVALS BY THERAPY (IN 1,029 PATIENTS WITH CARCINOMA OF THE BREAST)

	Operation Only				Operation plus Irradiation				Irradiation Only				All Therapies			
	No.	5 yr. survivals			No.	5 yr. survivals			No.	5 yr. survivals			Total No.	5 yr. survivals		
		No.	Per cent	Not coded		No.	Per cent	Not coded		No.	Per cent	Not coded		No.	Per cent	Not coded
LOCAL RECURRENCES	97	26	26	16	300	75	24	32	17	1	5	4	423	102	24.1	52
I. Local recurrence treated by operation or irradiation*	32	11	34	2	215	52	24	17	9	1	11	0	256	64	25.0	19
A. Metastases Discovered	26	7	26	1	205	45	21	17	8	1	12	0	239	53	22.1	18
(1) Metastases treated by irradiation	0	0	0	0	179	42	23	13	8	1	12	0	187	43	22.9	13
(2) Metastases not treated by irradiation	26	7	26	1	26	3	11	4	0	0	0	0	52	10	19.2	5
B. No Metastases Discovered	6	4	66	1	10	7	70	0	1	0	0	0	17	11	64.6	1
II. Local recurrence not treated by operation or irradiation	65	15	23	14	94	23	24	15	8	0	0	4	167	38	22.6	33
A. Metastases Discovered	60	13	21	13	83	18	21	14	8	0	0	4	151	31	20.5	31
(1) Metastases treated by irradiation	0	0	0	0	64	15	23	12	5	0	0	3	69	15	21.7	15
(2) Metastases not treated by irradiation	60	13	21	13	19	3	15	2	3	0	0	1	82	16	19.5	16
B. No Metastases Discovered	5	2	40	1	11	5	45	1	0	0	0	0	16	7	43.7	2
NO LOCAL RECURRENCES	265	154	58	69	248	105	42	42	93	5	5	57	606	264	43.5	168
A. Metastases Discovered	102	22	21	39	171	44	25	36	86	3	3	53	359	69	19.2	128
(1) Metastases treated by irradiation	0	0	0	0	112	33	29	23	64	1	15	42	176	34	19.2	65
(2) Metastases not treated by irradiation	102	22	21	39	59	11	18	13	22	2	9	11	183	35	19.1	63
B. No Metastases Discovered	163	132	80	30	77	61	79	6	7	2	25	4	247	195	78.9	40

* All local recurrences under "Operation Only" were treated by operation and all local recurrences or metastases in the remainder of the table were treated by irradiation.

Table XXIII

LOCAL RECURRENCES AND METASTASES—SUMMARY OF NUMBER AND PERCENTAGE TREATED AND PERCENTAGE 5 YEAR SURVIVALS

	Number	Percentage of Total Studied	Percentage of Subtotals Studied	Percentage 5 yr. Survivals
Total Coded Patients—Carcinoma of Breast	1,092	100.0		
LOCAL RECURRENCES	423	41.1	100.0	24.1
I. Local recurrence treated by operation or irradiation (32 operation and 224 irradiation)	256	24.8	60.5	25.0
A. Metastases Discovered	239	23.2	54.1	22.1
(1) Metastases treated by irradiation	187	18.1	44.2	22.9
(2) Metastases not treated by irradiation	52	5.0	12.2	19.2
B. No Metastases Discovered	17	1.6	4.0	64.6
II. Local recurrence not treated by operation or irration	167	16.2	39.4	22.6
A. Metastases Discovered	151	14.6	35.6	20.5
(1) Metastases treated by irradiation	69	6.7	16.3	21.7
(2) Metastases not treated by irradiation	82	7.4	19.3	19.5
B. No Metastases Discovered	16	1.5	3.7	43.7
NO LOCAL RECURRENCES	606	58.8	100.0	43.5
A. Metastases Discovered	359	34.8	59.2	19.2
(1) Metastases treated by irradiation	176	17.1	29.0	19.2
(2) Metastases not treated by irradiation	183	17.7	30.1	19.1
B. No Metastases Discovered	247	24.0	40.9	78.9

TABLE XXIV

COMPARISON OF 5 YEAR SURVIVAL RATES FOR PATIENTS TREATED AND NOT TREATED FOR DISTANT METASTASES (IN SERIES OF 1,029 PATIENTS WITH CARCINOMA OF BREAST)

	Number of Patients	5 Year Survival		Not Coded as to Survival* (number)	5 Year Survival if all patients not coded as to survival had lived (per cent)
		Number	Per cent		
Patients recorded as having metastases treated by irradiation	432	92	21.2	93	42.8
Patients recorded as having metastases not treated by irradiation	317	61	19.2	84	45.6
Patients recorded as having no metastases	280	213	76.0	43	91.4

* Survival or nonsurvival unknown, considered as dead.

423 patients, 256 were treated by operation or irradiation for the local recurrences, and 167 were not treated. Twenty-five per cent of those treated and 22.6 per cent of those *not* treated survived five years. Of the 256 patients treated for local recurrences, 239, or 93 per cent, had distant metastases, and of the 167 not treated for local recurrences, 151, or 90 per cent, had demonstrable distant metastases. It is of interest to note that in the 606 patients who did not develop local recurrences, there were 359 who developed distant metastases. We do not believe that this experience is unique.

Also in Table XXII, one observes that in each part of the table where patients developing metastases are recorded there is a slight improvement in five year survival rates for those treated for metastases as compared to those not treated. However, in patients having no local recurrences and no metastases, there is a higher five year survival rate; namely, 78.9 per cent.

Table XXIII is a summary of Table XXII. It will be noted that 423, or 41.1 per cent, of the 1,029 patients developed local recurrences, and 60.5 per cent of those with local recurrences were treated by operation or irradiation, with 25 per cent surviving for five years. As indicated, 606, or 58.8 per cent, of the 1,029 patients did *not* de-

TABLE XXV

TUMOR DOSAGE FOR LOCAL RECURRENCES AND/OR METASTASES WITH REFERENCE TO EFFECT ON THE LOCAL LESION BY VARIOUS QUANTITIES OF ROENTGEN RADIATION

Tumor Dosage	Totals*	No Change or Increase in Size	Slight Regression	Marked Regression	Disappearance of Tumor	Effect not Coded
50– 499 r	266	82	115	49	9	11
500–1,499 r	529	119	208	133	34	35
1,500–2,999 r	350	69	128	88	39	26
3,000–3,999 r	79	10	28	27	9	5
4,000–4,999 r	28	3	6	17	1	1
5,000–5,999 r	18	2	8	4	2	2
over 6,000 r	31	5	4	12	9	1
Totals	1,301	290	497	330	103	81

* The figures in this table refer to areas treated and not to the number of patients treated.

velop local recurrences, and of these 606, there was a 43.5 per cent five year survival. Survival in the group of 606 not developing local recurrences approximates the number not developing metastases.

Table XXIV is partly a summary of Tables XXII and XXIII and shows the relationship between the total five year survival of those treated or not treated for metastases. It indicates very little, if any, improvement in five year survival rate by radiation treatment of metastasis. However, it significantly shows a 76.0 per cent five year survival rate in the group of patients recorded as having no metastasis.

Table XXV presents the total number of local recurrences or metastases treated by roentgen therapy and indicates that 1,145 of the 1,301 areas treated received less than 3,000 tissue roentgens. In this group of 1,301, there were 290 that showed no change or increase in size, 497 had slight regression, 330 had marked regression, 103 had disappearance of tumor and 81 were not coded as to effect.

Included in the above group of 1,301 areas treated by roentgen therapy for local recurrence and/or metastasis were 350 areas in bones. This fact is not indicated in Table XXV, but it may be stated that the majority of bony skeleton regions treated were in the spine. The median or average tissue dose used was 1,500 r. In the 350 areas in bones the results were as follows: no change or increase in size 109, slight regression in 127, marked regression in 90, complete disappearance in 5 and 19 not coded as to effect.

Table XXVI shows that 76 local recurrences or metastases were treated with radium. In the majority of patients the radium was used locally in the form of a plaque containing radium needles or radium cells. In 64 instances the tumor dose was 3,000 r or less, and slight regression was noted in 24 after such applications.

Table XXVII indicates the number of patients and five year survivals in patients having irradiation for recurrences or metastases added to other forms of radiation therapy given prior thereto. It is to be noted that 469 of 667 patients had irradiation for recurrences or metastases.

INFLUENCE OF THE OVARIES ON BREAST CARCINOMA

Correlations were developed between certain grossly measurable functions of the ovaries as pre- or postmenopause, pregnancy or lactation, castration by surgery or irradiation, etc., and breast carcinoma. These correlations are presented in the form of tables of statistical data.

Table XXVIII shows that 336 patients had

TABLE XXVI

TUMOR DOSAGE FOR LOCAL RECURRENCES OR METASTASES WITH REFERENCE TO EFFECT ON THE LOCAL LESION BY VARIOUS QUANTITIES OF RADIATION FROM RADIUM

Tumor Dosage*	Totals†	No Change or Increase in Size	Slight Regression	Disappearance of Tumor or Nodes	Effect not Coded
50– 500 r	40	9	18	1	12
500–1,500 r	15	1	5	1	8
1,500–3,000 r	9	1	1	2	5
3,000–4,000 r	1	0	0	0	1
4,000–5,000 r	4	1	2	0	1
5,000–6,000 r	1	1	0	0	0
over 6,000 r	6	0	5	0	1
Totals	76	13	31	4	28

* In the majority of patients the radium was used locally in the form of a pack or plaque containing radium needles.
† The figures in this table refer to areas treated and not to the number of patients treated.

TABLE XXVII

NUMBER AND SURVIVAL OF PATIENTS HAVING IRRADIATION FOR RECURRENCES OR METASTASES ADDED TO OTHER FORMS OF RADIATION THERAPY GIVEN PRIOR THERETO

	Pre-operative Irradiation Only		Pre-operative and Post-operative Irradiation		Post-operative Irradiation Only		Post-operative Prophylactic Irradiation		Operation and Radiation for Recurrences and Metastases		Unclassified Radiation and Operation		Irradiation Only	
	No.	5 yr. Surv. No.	No.	5 yr. Surv. No.	No.	5 yr. Surv. No.	No.	5 yr. Surv. No.	No.	5 yr. Surv. No.	No.	5 yr. Surv. No.	No.	5 yr. Surv. No.
Total patients (667)	24	5	34	10	303	106	29	12	157	47	10	0	110	6
Number and survival of those in the above treated groups who had additional irradiation for recurrence and metastases (469)	16	0	20	6	170	36	27	11	157	47	1	0	78	2

not reached the menopause, 460 had reached the menopause, 214 did not have the menopausal history stated on the original case records, and 19 had no coding as to menopause on the I.B.M. code cards. In the group not having reached the menopause the five year survival rate was 5.7 per cent better than in those who had reached the menopause prior to discovery of breast carcinoma (39.8 as against 34.1 per cent).

Table XXIX includes 17 patients who had castration by roentgen therapy and 34 patients who had bilateral oophorectomies *before* the diagnosis of breast carcinoma was made. The total five year survival rate for these 51 patients was 33.3 per cent, which is slightly less than the gross five year survival of 35.5 per cent for the whole 1,029 patients studied.

Tables XXX and XXXI present a study of the 74 patients who had therapeutic castration. Seventy-one patients had irradiation and 3 had surgery for this purpose. It is not indicated in the two tables, but of the 71 patients having therapeutic castration by irradiation, 65 had it by means of roentgen therapy and 6 by means of radium. In the 65 having roentgen irradiation, the tissue roentgens averaged 1,500 r or less, delivered in ten days to one month for 59, and averaged 500 r delivered

TABLE XXVIII

NUMBER AND PERCENTAGE 5 YEAR SURVIVAL OF PATIENTS WHO WERE PRE- OR POST-MENOPAUSE WHEN BREAST CARCINOMA DEVELOPED

Classification as Regards Menopause	Total Cases	Operation Only			Operation plus Irradiation			Irradiation Only			All Therapies
		No. of cases	5 yr. survival		No. of cases	5 yr. survival		No. of cases	5 yr. survival		5 yr. survival
			No.	Per cent		No.	Per cent		No.	Per cent	Per cent
Menopause not reached	336	110	59	53.6	207	73	35.2	19	2	10.5	39.8
Menopause reached*	460	156	79	50.6	248	75	30.2	56	3	5.3	34.1
Menopause history not stated on records	214	91	41	45.0	89	32	35.9	34	1	2.9	34.5
Menopause history not coded	19	5	1	20.0	13	0	0	1	0	0	5.2
Totals	1,029	362	180	49.7	557	180	32.3	110	6	5.4	35.5

* Under "menopause reached" are included the 51 patients who had roentgen castration or bilateral oophorectomy before the diagnosis of breast carcinoma was made.

TABLE XXIX

NUMBER AND PERCENTAGE 5 YEAR SURVIVALS OF PATIENTS HAVING ROENTGEN OR SURGICAL CASTRATION BEFORE DIAGNOSIS WAS MADE OF CARCINOMA OF BREAST

	Number of Cases	5 Year Survivals	
		Number	Per cent
Roentgen castration	17	4	23.5
Bilateral oophorectomy	34	13	38.2
Totals	51*	17	33.3

* These patients are included in Table XXVIII as having reached the menopause before the diagnosis of carcinoma of breast was made.

in one to ten days for 6 patients.* In the 6 patients having castration by radium the tissue roentgens were 3,000 r or less for 4 patients, and were 5,000 r for 2 patients, in both instances being delivered in one to ten days. The total five year survival for patients so castrated was 33.7 per cent.

Table XXXVI indicates that 16 patients had a breast carcinoma discovered during pregnancy or lactation, 7 of these were during pregnancy and 9 during lactation. This table also shows that 3 of the 16 had operation alone and 13 had operation plus irradiation. The total five year survival was 7 out of the 16 patients.

Tables XXXIII and XXXIV demonstrate a comparison between the patients therapeutically castrated and those *not* castrated therapeutically with the cases classified respectively by Richards' Stage of Disease[4] and Portmann's Clinical Group Classification for Carcinoma of the Breast.[3] The total five year survival was 35.7 per cent for those in the series *not* castrated therapeutically and 33.7 per cent for those castrated therapeutically.

Table XXXV shows that in the 53 patients† having irradiation castration for recurrences or metastases there was in the

* The dosage referred to was thought to be adequate on the basis of interruption of the menstrual cycle. Today we employ the vaginal smear technique to indicate the presence or absence of estrogenic function following roentgen therapy. We believe it important to know whether or not there is such evidence of estrogenic function if one attempts to evaluate the effect of castration either by surgical or other methods.

† These 53 patients are included in the 74 having therapeutic castration, which are studied in Tables XXX and XXXI.

TABLE XXX

NUMBER OF 5 YEAR SURVIVALS OF PATIENTS HAVING VARIOUS FORMS OF CASTRATION THERAPEUTICALLY (RICHARDS' STAGE OF DISEASE CLASSIFICATION FOR CARCINOMA OF BREAST)

Castration	Number Cases	Stage I		Stage II		Stage III		Stage IV		Stage V		Stage not Coded		All Stages
		Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	5 yr. surv. Per cent
I. Primary Prophylactic														
A. Irradiation	3	0	0	0	0	0	0	1	1	2	0	0	0	33.0
B. Operation	0	0	0	0	0	0	0	0	0	0	0	0	0	0
II. Preoperative Prophylactic														
A. Irradiation	1	0	0	0	0	0	0	1	1	0	0	0	0	100.0
B. Operation	1	0	0	0	0	1	0	0	0	0	0	0	0	0
III. Postoperaitve Prophylactic														
A. Irradiation	14	1	1	4	2	8	1	1	1	0	0	0	0	35.0
B. Operation	0	0	0	0	0	0	0	0	0	0	0	0	0	0
IV. Castration for Recurrence or Metastases														
A. Irradiation	53	8	4	14	9	11	4	11	1	7	0	2	0	33.0
B. Operation	2	0	0	1	0	0	0	0	0	0	0	1	0	0
V. Totals														
A. Irradiation	71	9	5	18	11	19	5	14	4	9	0	2	0	35.0
B. Operation	3	0	0	1	0	1	0	0	0	0	0	1	0	0
GRAND TOTALS	74	9	5	19	11	20	5	14	4	9	0	3	0	33.7

TABLE XXXI

NUMBER OF 5 YEAR SURVIVALS OF PATIENTS HAVING VARIOUS FORMS OF CASTRATION THERAPEUTICALLY (PORTMANN'S GROUP CLASSIFICATION FOR CARCINOMA OF BREAST)

Castration	Number of Cases	Group I		Group II		Group III		Group IV		Group not Coded		All Stages
		Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	5 yr. surv. Per cent
I. Primary Prophylactic												
A. Irradiation	3	0	0	1	1	1	0	1	0	0	0	33.0
B. Operation	0	0	0	0	0	0	0	0	0	0	0	0
II. Preoperative Prophylactic												
A. Irradiation	1	0	0	1	1	0	0	0	0	0	0	100.0
B. Operation	1	0	0	1	0	0	0	0	0	0	0	0
III. Postoperative Prophylactic												
A. Irradiation	14	5	4	4	1	1	0	0	0	4	0	35.0
B. Operation	0	0	0	0	0	0	0	0	0	0	0	0
IV. Castration for Recurrence or Metastases												
A. Irradiation	53	29	12	15	5	4	0	3	0	2	1	33.0
B. Operation	2	1	0	0	0	1	0	0	0	0	0	0
V. Totals												
A. Irradiation	71	34	16	21	8	6	0	4	0	6	1	35.0
B. Operation	3	1	0	1	0	1	0	0	0	0	0	0
GRAND TOTALS	74	35	16	22	8	7	0	4	0	6	1	33.7

local lesion no change or increase in size in 22, slight regression in 7, marked regression in 8, complete disappearance in 3 and no recorded effect in 13 patients.

SIZE OF ORIGINAL TUMOR OF BREAST

It is believed by many that the prognosis becomes less favorable with increasing size of the carcinoma in the breast. In order to test this observation in this series of 1,029 patients, three tables were prepared from our data.

Table XXXVI correlates size of the breast tumor with the general type of therapy.

TABLE XXXII

NUMBER AND 5 YEAR SURVIVAL OF PATIENTS DEVELOPING CARCINOMA OF THE BREAST DURING PREGNANCY OR LACTATION BY TYPE OF THERAPY BY RICHARDS' STAGE OF DISEASE CLASSIFICATION

Classification Stage	Number of Cases	Operation Only				Operation plus Irradiation			
		Simple mastectomy		Radical mastectomy		Simple mastectomy		Radical mastectomy	
		Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	Cases	5 yr. surv. No.	Cases	5 yr. surv. No.
I	1	0	0	0	0	1	1	0	0
II	4	0	0	1	0	0	0	3	2
III	7	1	1	0	0	3	1	3	0
IV	2	1	1	0	0	0	0	1	0
V	1	0	0	0	0	1	0	0	0
Not coded	1	0	0	0	0	0	0	1	1
Totals	16*	2	2	1	0	5	2	8	3

* Seven cases discovered during pregnancy and 9 during lactation.

TABLE XXXIII

EFFECT OF CASTRATION OR NONCASTRATION ON 5 YEAR SURVIVALS. CASES CLASSIFIED ACCORDING TO RICHARDS' STAGE OF DISEASE CLASSIFICATION CARCINOMA OF BREAST

Classification Stage	Total No. of cases	Not Castrated (Therapeutic)			Castrated (Therapeutic)		
		Number	5 year survival		Number	5 year survival	
			No.	Per cent		No.	Per cent
I	122	113	91	80.5	9	5	55.5
II	170	151	70	46.2	19	11	57.8
III	314	294	128	43.5	20	5	25.0
IV	244	230	45	19.5	14	4	28.6
V	137	128	3	2.3	9	0	0
Not coded	42	39	4	10.2	3	0	0
Totals	1,029	955	341	35.7	74	25	33.7

The table shows: (1) that under the heading "Operation Only" the majority of tumors were smaller than under the headings "Operation plus Irradiation" or "Irradiation Only;" (2) that the five year survival decreases directly as the size of tumor increases, and (3) that the presence of a small tumor is no sure guarantee of survival regardless of therapy used.

Table XXXVII correlates size of the breast tumor with the type of radiation therapy used plus operation. The table shows as does Table XXVI that five year survival decreases directly as the size of tumor increases.

Table XXXVIII correlates size of the breast tumor with the patient's statement of duration of symptoms to date of examination in this or other hospitals. These statements regarding symptoms may be grossly inaccurate, but if one could assume that they were even, in part, correct, one will observe a great difference in survival throughout the table. Even casual examination of the data suggests: (1) there is a great difference in growth rates of tumors of the breast, and (2) one should use growth rate as well as size in estimating prognosis.

COMMENT

In considering the treatment of local recurrences there is a gain of 2.4 per cent

TABLE XXXIV

EFFECT OF CASTATION OR NONCASTRATION ON 5 YEAR SURVIVALS. CASES CLASSIFIED ACCORDING TO PORTMANN'S CLINICAL GROUP CLASSIFICATION CARCINOMA OF BREAST

Classification Group	Total No. of Cases	Not Castrated Therapeutically			Castrated Therapeutically		
		Number	5 year survival		Number	5 year survival	
			No.	Per cent		No.	Per cent
I	490	455	248	54.5	35	16	45.7
II	290	268	66	24.2	22	8	36.8
III	95	88	20	22.7	7	0	0
IV	115	111	3	2.7	4	0	0
Not coded	39	33	4	12.1	6	1	16.6
Totals	1,029	955	341	35.7	74	25	33.7

in five year survival as compared to the absence of treatment for local recurrences. No significant reduction in metastases was observed by treating the local recurrences. Of the patients having no local recurrences there was metastasis in 59.1 per cent. Patients having radiation treatment of metastatic disease showed a 2 per cent increase in five year survival as compared to those not treated.

An evaluation of the influence of ovarian function on breast carcinoma indicated that there was a slightly better five year survival in those not having reached the

TABLE XXXV

IRRADIATION CASTRATION FOR RECURRENCES AND/OR METASTASES—EFFECT ON LOCAL LESIONS

Effect on Local Lesions	Castrated Patients
No change or increase in size	22
Slight regression	7
Marked regression	8
Complete disappearance	3
Effect not recorded on patient's history	13
Total	53

TABLE XXXVI

NUMBER AND PER CENT OF 5 YEAR SURVIVALS DEPENDING ON SIZE OF ORIGINAL TUMOR OF BREAST AND TYPE OF THERAPY

Size of Tumor	Total Cases	Operation Only			Operation plus Irradiation			Irradiation Only			All Therapies	Survival or Nonsurvival Unknown, Considered as Dead		
Greatest Diameter*		Total No.	5 yr. survival		Total No.	5 yr. survival		Total No.	5 yr. survival		5 yr. survival	(No. of Cases)		
			No.	Per cent		No.	Per cent		No.	Per cent	Per cent	Operation	Op. & Irradiation	Irradiation
Under 20 mm.	205	96	69	71.8	106	44	41.5	3	1	33.3	55.5	14	8	0
20 to 49 mm.	319	118	66	55.9	176	65	36.9	25	2	4.0	41.7	20	17	10
50 to 89 mm.	279	86	24	27.9	158	42	26.5	35	2	5.7	24.3	21	32	17
90 mm. or over	92	28	4	14.2	39	4	10.2	25	0	0	8.6	19	9	19
Not stated	85	24	114	58.2	50	17	34.0	11	1	9.0	3.7	5	6	7
Not coded	49	10	3	30.0	28	8	28.6	11	0	0	22.4	6	2	8
Totals	1,029	362	180	49.7	557	180	32.3	110	6	5.4	35.5	85	74	61

* These measurements are clinical and not on the gross pathology specimen.

TABLE XXXVII

NUMBER AND PER CENT OF 5 YEAR SURVIVALS DEPENDING ON SIZE OF ORIGINAL TUMOR OF BREAST AND TYPE OF RADIATION THERAPY COMBINED WITH OPERATION

Size of Tumor	Number of Cases	Preoperative Irradiation Only*			Preoperative and Postoperative Irradiation†			Postoperative Irradiation Only‡			Postoperative Prophylactic Irradiation§			Operation and Irradiation for Recurrences or Metastases‖			Unclassified Irradiation and Operation¶			Survival or Nonsurvival Unknown, Considered as Dead					
Greatest Diameter		No.	5 yr. surv.		No.	5 yr. surv.		No.	5 yr. surv.		No.	5 yr. surv.		No.	5 yr. surv.		No.	5 yr. surv.		*	†	‡	§	‖	¶
			No.	%		No.	%		No.	%		No.	%		No.	%		No.	%	(Number of cases)					
Under 20 mm.	106	1	1	100	6	4	66	60	25	41	4	1	25	34	13	38	1	0	0	0	0	5	1	2	0
20 to 49 mm.	176	8	2	25	10	3	30	94	39	41	10	5	50	54	16	29	0	0	0	3	1	9	1	3	0
50 to 89 mm.	158	8	1	12	11	2	18	96	29	30	4	1	25	38	9	23	1	0	0	2	3	16	4	7	0
90 mm. or over	39	4	1	25	2	0	0	18	2	11	4	0	0	11	1	9	0	0	0	0	0	3	2	4	0
Not stated	50	1	0	0	2	1	50	27	8	29	5	3	60	15	5	33	0	0	0	0	0	3	0	3	0
Not coded	28	2	0	0	3	0	0	8	3	27	2	2	100	5	3	60	8	0	0	0	1	1	0	0	0
TOTALS	557	24	5	20	34	10	29	303	106	34	29	12	41	157	47	29	10	0	0	5	5	37	8	19	0

Note: These measurements are clinical and not on the gross pathology specimen.

TABLE XXXVIII

NUMBER OF 5 YEAR SURVIVALS DEPENDING ON SIZE OF BREAST TUMOR AND DURATION OF SYMPTOMS

Size of Tumor	Totals		Patient's Statement of Duration of Symptoms to Date of Examination in This or Other Hospitals																	
			Under 2 weeks		2 weeks to 1 month		1 to 2 months		3 to 5 months		6 to 11 months		12 to 23 months		24 to 35 months		36 months and over		Duration unknown	
Greatest Diameter	No. of pts.	No. 5 yr. surv.	No.	5 yr. surv. No.	No.	5 yr. surv. No.	No.	5 yr. surv. No.	No.	5 yr. surv. No.	No.	5 yr. surv. No.	No.	5 yr. surv. No.	No.	5 yr. surv. No.	No.	5 yr. surv. No.	No.	5 yr. surv. No.
Under 20 mm.	205	114	37	22	27	16	32	17	33	17	31	18	15	7	5	2	16	8	9	7
20 to 49 mm.	319	133	29	20	32	15	53	20	50	25	52	20	39	15	18	8	27	6	19	4
50 to 89 mm.	279	68	9	2	15	4	43	14	55	10	47	8	43	7	21	7	25	10	21	6
90 mm. or over	92	8	1	0	3	0	5	1	14	0	19	3	21	1	8	2	17	1	4	0
Not stated or coded	134	43	6	3	7	2	15	4	17	4	16	5	18	6	12	4	15	4	28	11
TOTALS	1,029	366*	82	47	84	37	148	56	169	56	165	54	136	36	64	23	100	29	81	28

* In considering this survival figure, it must be stated that there were 220 patients of the total series of 1,029 whose survival or nonsurvival was unknown and they were considered as dead for statistical purposes.

menopause. Carcinoma of the breast developed in 34 patients after bilateral oophorectomy.

In this series the use of castration therapeutically did not increase five year survival. Carcinoma developing in the breast during pregnancy or lactation, much to our surprise, had a survival rate which was surprisingly high. This observation will continue to be checked. Castration used for effect on local recurrences or metastases showed a beneficial effect in 18 out of 53 patients.

The size of the original tumor of the breast had a general relationship to prognosis—the larger the tumor, the worse the outlook. However, this is not strictly true and must be modified by consideration of the growth rate of the tumor as judged by duration of the tumor.

SUMMARY

1. In the series of 1,034 patients with carcinoma of the breast seen at the Hospital of the University of Pennsylvania, between 1923 and 1943, of which 1,029 cases were coded, there were 423 who had local recurrences, of which number 390 either had concurrent or later metastases. Of the 606 not having local recurrences, 359 had concurrent or later metastases.

2. The 256 patients having local recurrences treated by surgery or irradiation had a 25.0 per cent five year survival. The 167 patients having local recurrences not treated by surgery or irradiation had a 22.6 per cent five year survival.

3. The 432 patients recorded as having metastases treated by irradiation had a 21.2 per cent five year survival, while the 317 patients recorded as having metastases not treated by irradiation had a 19.2 per cent five year survival.

4. The tumor dose used for local recurrences and/or metastases with roentgen rays or radium was in most instances less than 3,000 r. The average effect of treatment was slight to marked regression of the local lesion.

5. The 280 patients recorded as not having metastases had a 76.0 per cent five year survival.

6. The 336 patients not having reached the menopause had a 39.8 per cent five year survival, and the 460 having reached the menopause naturally or by castration had a 34.1 per cent five year survival. The 214 patients not having menopausal history recorded had a 34.5 per cent five year survival.

7. The 74 patients having castration performed after the diagnosis of breast cancer showed a 33.7 per cent five year survival. In 53 castrated for recurrences or metastases, there was a beneficial effect on the local lesion in 18 patients.

8. In considering size of original tumor of the breast, 205 patients had a tumor under 20 mm., 319 between 20 to 49 mm., 279 between 50 to 89 mm., 92, 90 mm. or

over, and 134 did not have size stated on their records. The larger the tumor, the worse the prognosis.

9. In considering size of breast tumor with relation to patient's statement of duration, 82 patients stated that they knew of the tumor less than two weeks, yet only 57.3 per cent of these survived five years. There were 205 patients who had a tumor under 20 mm., yet only 55.6 per cent of these survived five years.

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NECROSIS OF THE BRAIN FOLLOWING ROENTGEN IRRADIATION*†

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THERE are few cases in the literature of necrosis of human brain following roentgen irradiation.[1,5,6,7] Some of these are of questionable validity because of the presence of co-existing pathological processes, such as brain tumor, intracranial infection, or hydrocephalus. Recently, Lowenberg-Scharenberg and Bassett[3] have reported 2 cases of degeneration of the brain following roentgen treatment of suspected carcinoma of the scalp. This is a report of a similar case.

Our objective in reporting the present case is to call the attention of neurosurgeons and neuropathologists to a postirradiation picture which may simulate glioma; it is also to caution the radiation therapist against the danger of high intensity irradiation over a relatively short period of time, when a fair volume of tissue is irradiated. It is far from the authors' intention to discourage anyone from using irradiation properly when indicated in the treatment of diseases of the central nervous system.

CASE REPORT

C.T. The patient was a white male tailor, aged fifty-four. In November, 1949, a skin lesion above the left ear was diagnosed "squamous cell carcinoma" but not verified histopathologically. At another hospital, over a period of ten days, 4,800 roentgens were given to the lesion. This consisted of a daily treatment of 480 r at 150 kv. through a 5 cm. portal at a 25 cm. distance (filter 0.5 mm. copper and 0.5 mm. aluminum). Following this treatment the lesion healed.

In early February, 1952, approximately twenty-six months after irradiation, the patient became confused and forgetful. A few days later he complained of bifrontal headaches and dizziness. He was admitted with these symptoms on February 20, 1952.

Examination. The patient was drowsy, disorientated, and dysphasic. There was a right hemiparesis, right hemihypesthesia, and right homonymous hemianopsia. Above the left ear the skin was atrophic, dry, crusted, and scaling.

Ventriculography showed a space-occupying lesion inferior and lateral to the temporal horn of the left lateral ventricle.

Course. A left temporoparietal osteoplastic craniotomy was done on February 22, 1952. The second left temporal convolution, 5 cm. from the tip of the temporal lobe, was widened, semitranslucent, and discolored greenish-yellow. This area was firm, resilient, and well demarcated. Grossly, this appeared to be a glioma and a large block of tissue was resected.

After an uneventful course the patient was discharged on the nineteenth postoperative day. At this time considerable improvement had occurred but some confusion, dysphasia, and a minimal right hemiparesis remained.

Histological Pathology. In the microscopic sections degenerating and necrotic gray and white matter were recognized. Although both regions contained such areas the more severe injury was in the white matter. Here, focal areas were almost completely necrotic containing cellular debris, leukocytes, and fat (Fig. 1). These foci usually surrounded a vessel.

Although some neurones survived, the cortical cytoarchitecture was destroyed. Most of the remaining neurones were abnormal, having irregular margins, tortuous dendrites, and dark marginated nuclei (Fig. 2).

In several areas the leptomeninges were thickened by a collagenous fibrosis. Discrete groups of small vessels were observed, which extended from the leptomeninges into the brain for a short distance and resembled telangiectases.

The vessels everywhere had undergone some alteration, often striking in amount. The walls

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† Aided by a grant from The Cancer Research Society, Inc., Montreal.

FIG. 1. Focus of necrosis in white matter containing cell debris and leukocytes (approximately ×21).

of several consisted of an irregular wide band of hyaline material while others were composed of many concentric strands of the same hyaline substance. Around other vessels there were dense cuffs of lymphocytes (Fig. 3) and an extensive collagenous fibrosis which frequently was vascularized (Fig. 4). A number of vessels were necrotic and could scarcely be distinguished from the surrounding tissue. Along with the perivascular fibrosis there had been an endarteritis. In all of the changed vessels the lumen was closed or, at least, considerably narrowed (Fig. 5).

The gold chloride sublimate and silver carbonate preparations showed an intense gliosis around the areas of degeneration (Fig. 6). Those astrocytes close to the necrotic areas were swollen and their processes fragmented (Fig. 7).

Comment. The pathological observations in this case suggest that the parenchyma of the brain is resistant to intensive roentgen irradiation but that the vessels react as they do in other tissues. A periarteritis and an endarteritis with partial or complete vascular occlusion were found. The injury caused by this focal ischemia was much more severe in the white matter than in the cortex. This relative sparing of the cortex supports the suggestion that the primary radiation injury is vascular. The blood supply to the cortex is much greater than to the white matter, rendering the former less liable to damage than the latter from chronic progressive ischemia.

It is not known to what extent neurones are resistant to radiation injury. Their presence solely in the gray matter cannot account for the less severe necrosis there. This is borne out by the fate of the astrocytes which are common to both gray and white matter and which, therefore, may be used as a basis for comparison of the path-

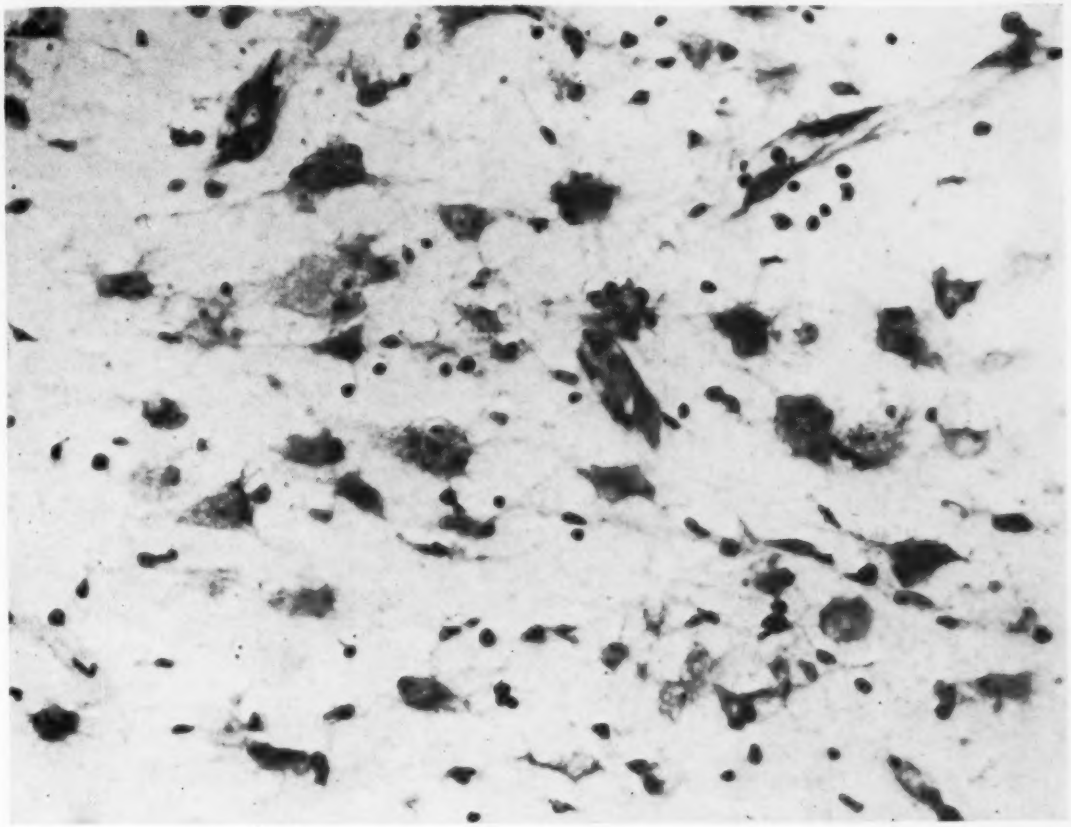

Fig. 2. Abnormal neurones with irregularity of shape, chromatolysis, and pyknosis (approximately ×180).

ological processes existing in these areas. In the cortex many of these cells were viable and reactive while in the white matter almost all of them were destroyed.

Symptoms do not occur until months or years after irradiation. In this case twenty-six months elapsed but in other reported cases the interval has been even longer.[2,4] This does not mean that vascular alterations do not begin early after irradiation but rather that the vessel reaction is slowly progressive and a significant degree of ischemia is not reached until many months have passed.

The late radiation effects observed and reported in this case should not be surprising if one considers the high intensity of the dose administered in relationship to the time factor. A total dose of 4,800 r, measured in air, represents, with backscatter, a surface dose estimated at approximately 5,500 r; such an amount administered in only ten days is a large dose over an area of a few square centimeters. It becomes practically excessive when delivered to a surface area of more than 25 sq. cm. It must also be realized that with the quality of roentgen radiation utilized here, approximately 50 per cent of the radiant energy will be absorbed by the tissue within a depth of 5 cm. This high absorption rate of 2,750 r in ten days in a cylinder of nervous tissue, measuring 5 cm. in diameter and 5 cm. in depth, would give enough dissipation of radiant energy to induce permanent changes and damage to the blood vessels and, secondarily, to the glial cells and perhaps the neurones. In other words, the permanent damage to intracerebral blood vessels may be the most significant factor in relationship to gradual late alteration in the structure of

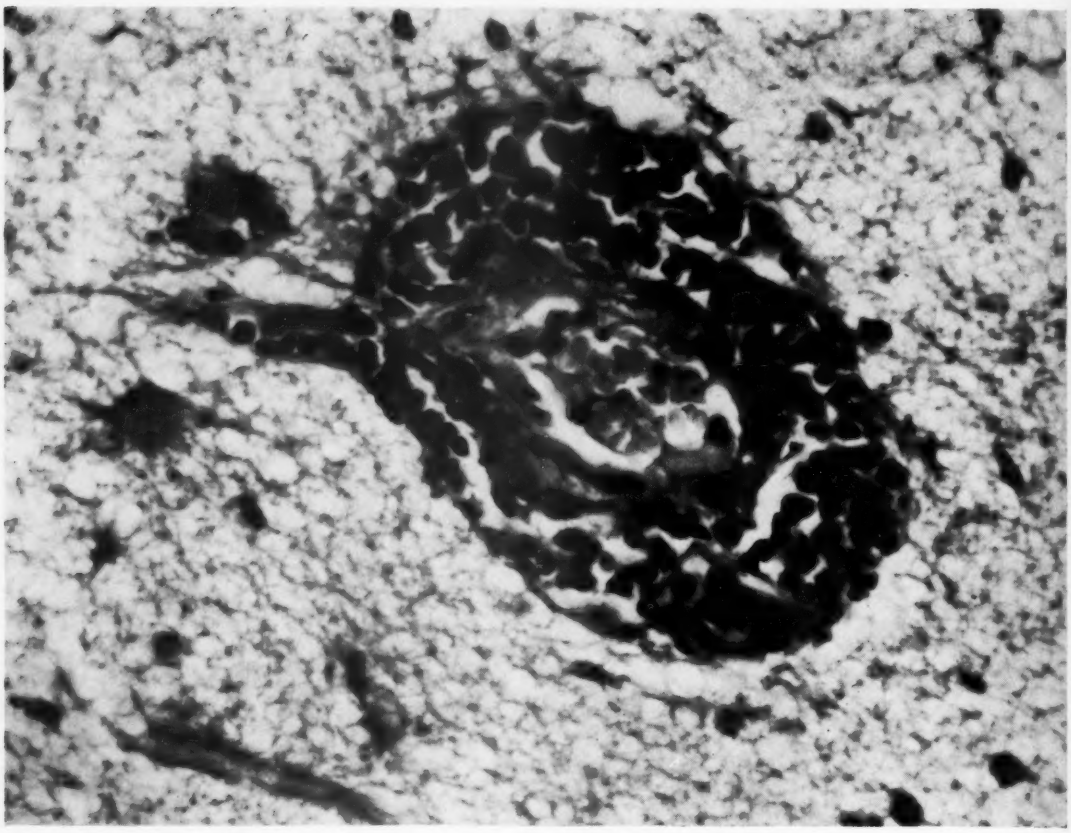

FIG. 3. Vessel with a cuff of lymphocytes (approximately ×180).

the glial cells and especially the astrocytes.

The postirradiation damage to the brain, reported in this case, stands in marked contradistinction to the experience of one of the authors and other associates, in the treatment with roentgen irradiation of intracranial neoplasms. At the Montreal Neurological Institute and the Department of Radiology, Royal Victoria Hospital, in the treatment of pituitary tumors, a large number of normal temporal lobes have been irradiated with larger doses than that utilized in the case reported here. The surface area treated has been routinely of approximately the same size, between 20 to 25 sq. cm. The skin dose ordinarily ranges up to 5,000 r and even more. The minimal depth dose through the temporal lobes down to the level of the pituitary gland has often been of the order of 5,000 r minimum over an average time distribution of forty-five days. Yet, long term follow-ups in a group of more than 50 cases of pituitary tumors have not brought to light late skin reactions comparable to the amount of skin atrophy shown by this patient. During clinical follow-up, or at time of subsequent operations, not a single case of brain damage has been suspected in the same series of cases. It is believed that the most important reason why skin and brain damage apparently have not been induced is the distribution in time over a period four to five times longer than in the present case of skin and brain damage.

Further, in the management of brain tumors with roentgen therapy, administering depth doses more than twice as great as in the present case, we have not yet found immediate or late evidence of postirradiation cerebral damage.

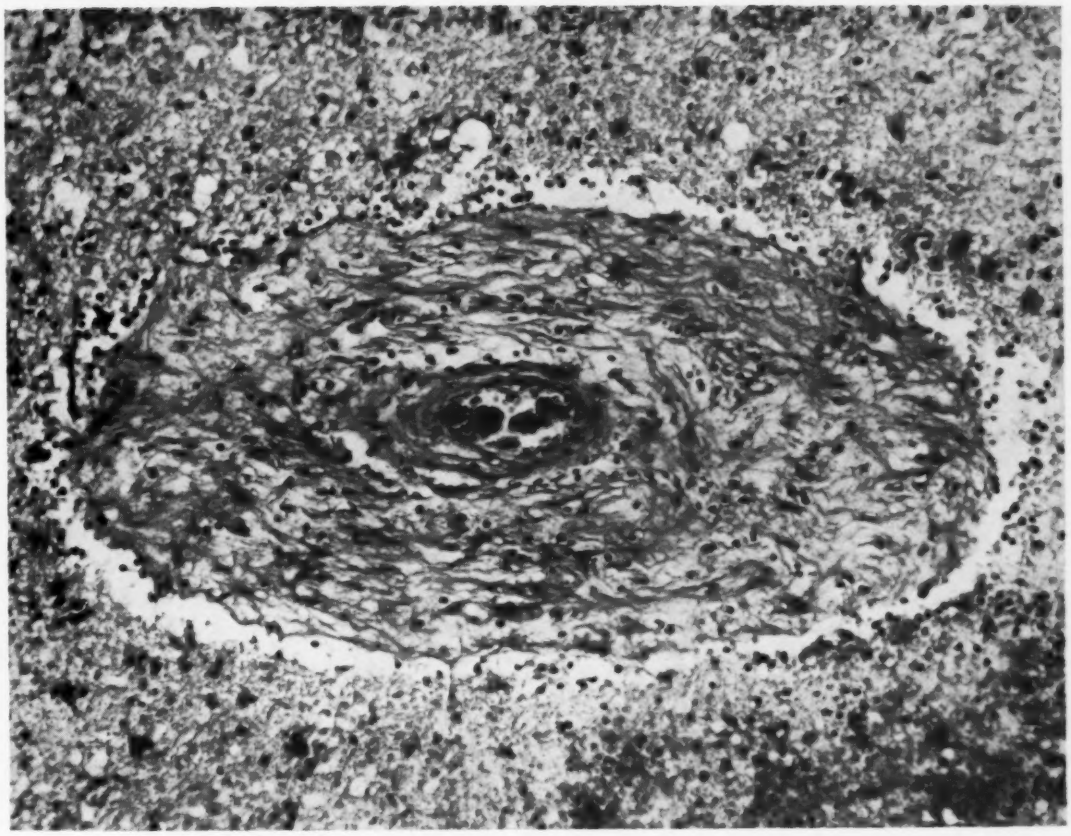

FIG. 4. Perivascular fibrosis around a narrowed vessel (approximately ×120).

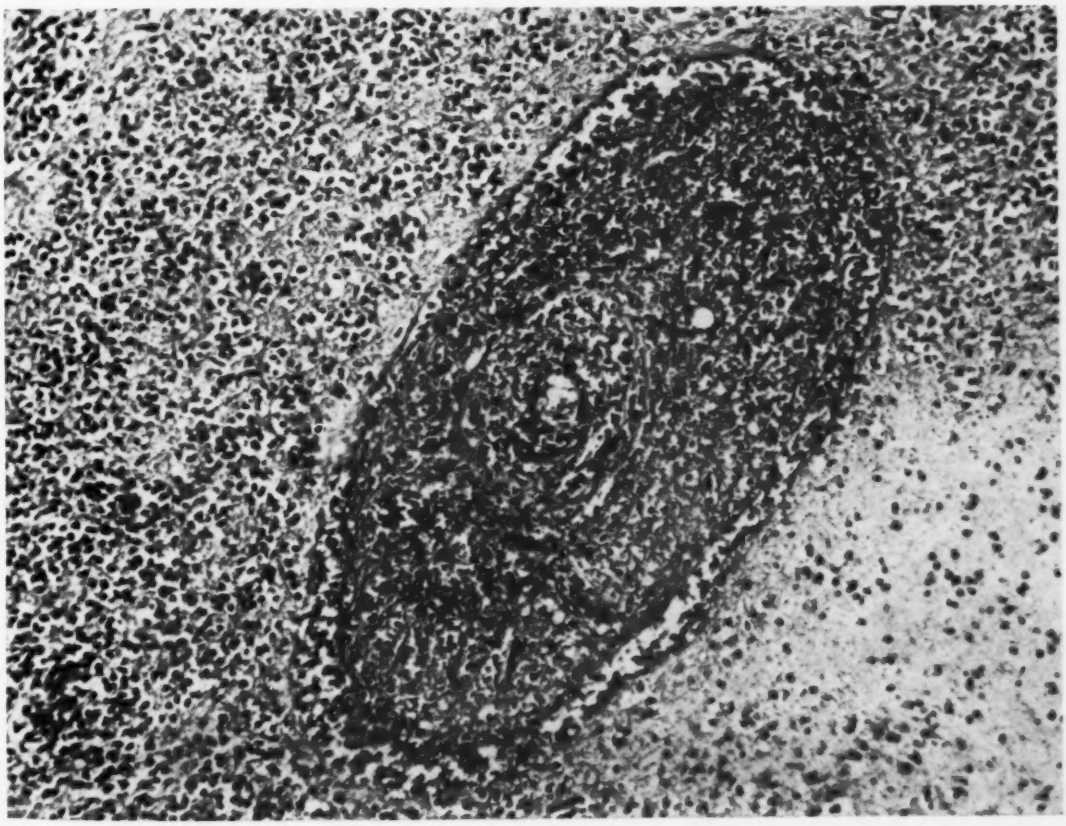

FIG. 5. Narrowed vessel with hyalinized wall and leukocytic infiltration (approximately ×120).

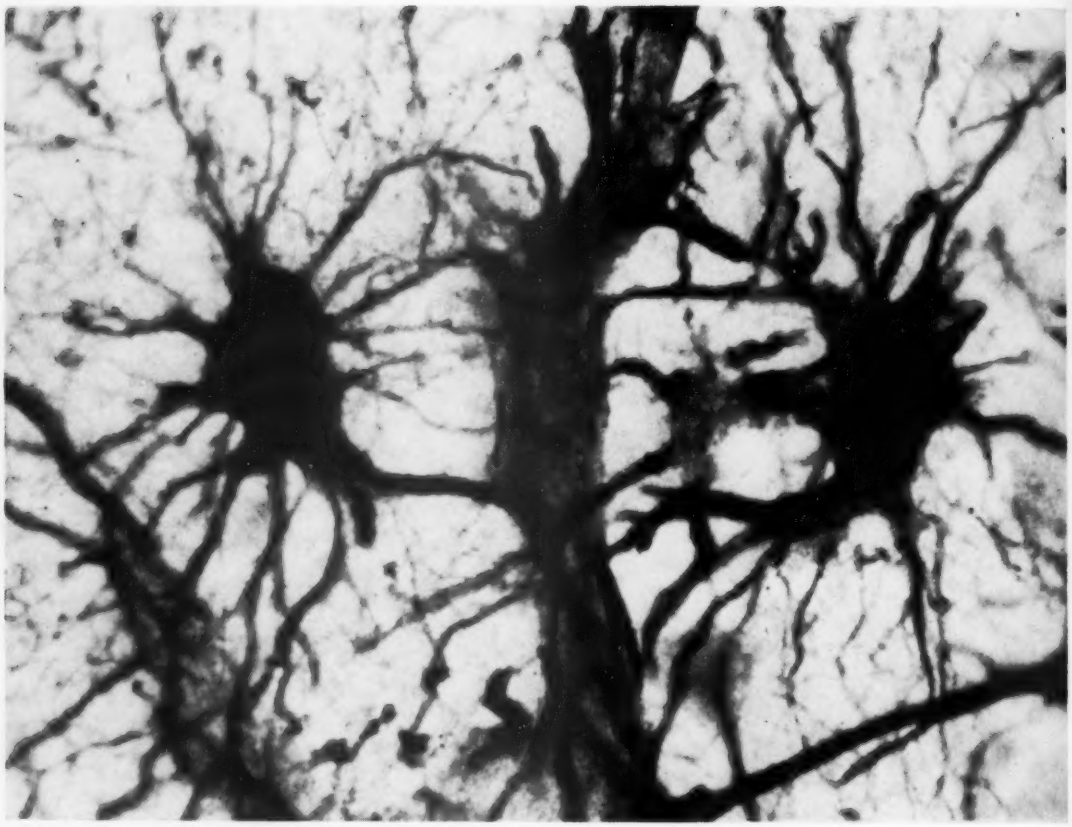

FIG. 6. Reactive gliosis in cortex (approximately ×400).

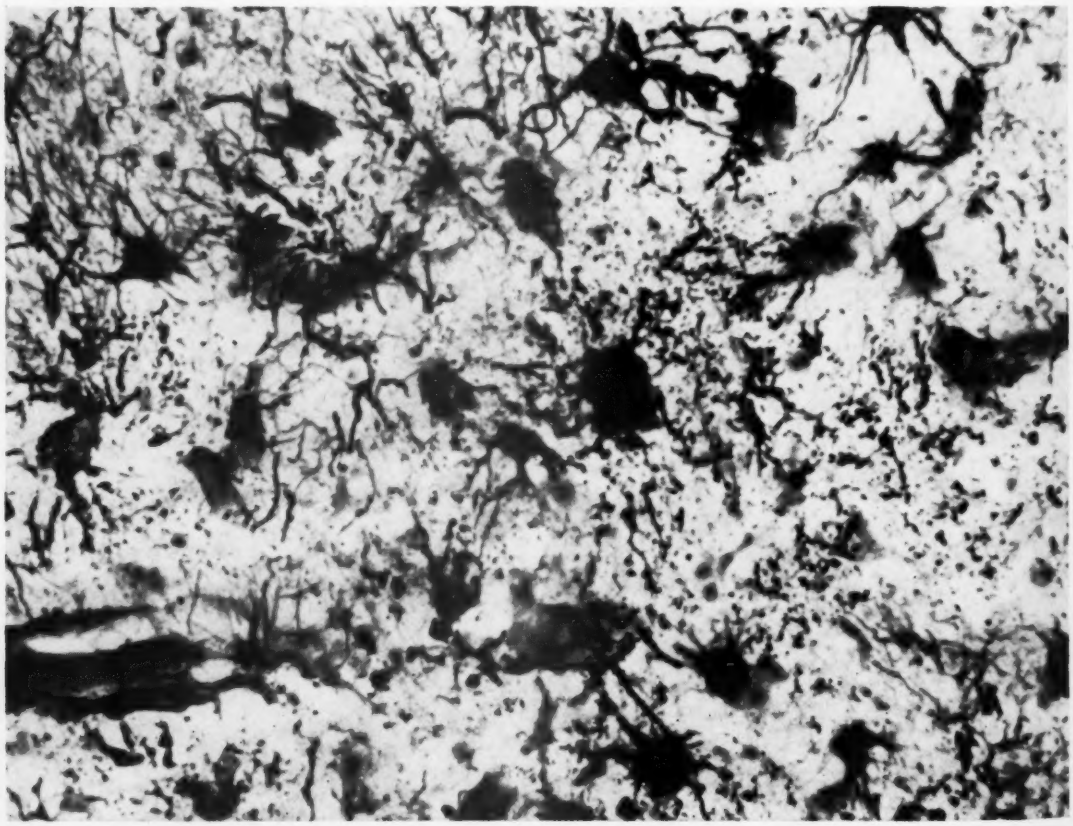

FIG. 7. Fragmentation of astrocytes at border of a necrotic area (approximately ×180).

SUMMARY

1. A case of postirradiation necrosis clinically simulating neoplasm is reported.

2. Focal degeneration and necrosis secondary to vascular occlusion from periarteritis and endarteritis were observed.

3. This degeneration was greater in the white matter than in the cortex, probably because of the latter's more abundant blood supply.

4. Ionizing radiations from radioactive material or from a roentgen tube are always likely to damage central nervous system structures if administered with high intensity in a relatively short period of time.

5. The most important factor in planning radiation therapy which will avoid unnecessary and unjustifiable postirradiation injury to the central nervous tissue lies in protraction of the dose over an adequate period of time.

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We wish to thank Dr. W. V. Cone and Dr. A. R. Elvidge for assistance in reporting this case.

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DISCUSSION

Dr. Joseph P. Evans, Cincinnati, Ohio. It seems to me that we all should be grateful to Dr. Dugger and his associates for this contribution reiterating the importance of a little recognized condition which emphasizes the need of very careful clinical follow-up of patients who have received radiation treatment. I think the fact that we see few such cases suggests the possibility that we have not been looking for them carefully enough.

The parallel to excessive radiation effects in pulmonary diseases is striking when one considers the basic pathology which affects the different tissues in these two situations. The changes in the blood vessels which have been so beautifully described here, and the changes in parenchyma, are parallel, apparently, to those which are seen in pulmonary disease.

Because I have either not been wise enough to recognize this condition in our own experience, or because the type of therapy which our radiologists use has been such as not to produce a change of this sort, my own experience is limited. However, I have had the opportunity of reviewing the manuscript of a paper from the Children's Medical Center in Boston which was presented at the Harvey Cushing Society meeting last spring by Dr. McLaurin, Dr. Bailey, Dr. Harsh and Dr. Focaut. This was an experimental study on the effects of radiation, both roentgen and gamma, on the spinal cord of animals, and it bears particularly on this problem. I am, with the permission of Dr. McLaurin, taking the liberty of reading from their description the histopathological changes which were studied by Dr. Bailey, their neuropathologist:

"The histologically identifiable damage clearly depended on two factors—the length of survival after irradiation, and the total amount of radiation received. The former was the more important of the two, indicating the progressive nature of the reaction.

"In the spinal cords of monkeys receiving the largest doses of radiation and allowed to survive for several months, total necrosis and disruption of both white and gray matter was found. The spinal arteries in the fibrosed and hyalinized meninges presented extensive intimal proliferation which in some instances progressed to complete occlusion. The intramedullary lesions could thus be regarded as a transverse infarction at the level of irradiation.

"The pathogenesis of the arterial change was followed by comparing the findings in several monkeys

of the series. At the earliest stages after irradiation the arteries appeared essentially normal. Later there was necrosis of the vessel walls and inflammatory cellular infiltration. This state was followed by healing and development of occlusive endothelial proliferation. At successive levels cephalad and caudad to the region of transverse infarction, the vascular changes became progressively less pronounced.

"The tissues of the spinal cord showed greater injury to the white matter than the gray. There was a marked glial proliferation in the areas of damage to the white matter. Below the area of maximum injury, some of the changes were undoubtedly due to retrograde degeneration, but the similarity of many of them to changes above the site of irradiation suggested that a direct radiation effect was also present.

"In contrast with the picture described above, the monkeys receiving equivalent dosages of radiation and sacrificed a few days later presented a relatively normal appearance. In the white matter a few myelin sheaths were swollen and partly degenerated. No morphological change was recognized in the gray matter.

"From the material it appeared that the lesions resulting in paraplegia were primarily vascular in origin, but that injury to neurones extended for considerable distances beyond the area of maximum irradiation. The white matter was more affected than the gray. The extent and distribution of the lesions were considered an indication that gamma and roentgen radiation can injure the nerve fibers directly without mediation of vascular lesions.

"Since the histological changes were greatly increased in severity with time, the injury was a continuing phenomenon, or at least one which was progressively more unmasked histologically with time."

So, in this experimental work I think we have a confirmation of the clinical work which was so beautifully demonstrated histologically by Dr. Dugger and his associates.

It is evident that all of the clinical studies to date, and these experimental studies, agree on the importance of the time factor; and another factor in this particular case, which is rather beyond my certain comprehension, is the question of backscatter in view of the type of filter that was used.

The matter deserves additional attention at this time, of course, as newer radiation methods are being developed. Whether these will be subject to the same limitations as the roentgen effects, as far as time is concerned, remains to be demonstrated.

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TREATMENT PLANNING IN ROENTGEN THERAPY*

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THE aim of radiotherapy in the treatment of any malignant growth is the uniform irradiation of a minimum but adequate volume of tissue to a dose that is likely to cause complete destruction of the growth. The volume of tissue treated should contain the malignant process and its probable extensions. The dosage should be uniform throughout to insure adequate irradiation of the entire tumor. The irradiation of any other tissue must be regarded as unnecessary and should be avoided wherever possible.

In the roentgen treatment of skin carcinoma, these conditions can be readily satisfied. Roentgen therapy of a malignant process below the surface of the body presents problems that cannot be as easily solved. The radiation must traverse skin and other normal tissues lying between the surface and the tumor. Furthermore, to achieve satisfactory dosage in the tumor, it is usually necessary to use more than one treatment beam, each of which contributes its quota of radiation. This results in unavoidable exposure of normal tissue. A treatment plan for a deeply situated tumor must represent a compromise between the maximal irradiation of the tumor and the minimal exposure of normal tissue.

In order to obtain the best compromise, calculation of the radiation distribution in the patient is an essential part of such a treatment plan. This computation should give a complete picture of the radiation distribution within the patient, except perhaps in the case of simple techniques requiring the use of single ports, or extreme palliative procedures. This is necessary for several reasons. Overdosage of sensitive areas or local regions can be prevented. Complete knowledge of the radiation distribution will help to determine whether recurrence is due to inadequate irradiation and this will be of value in re-treatment. The accurate comparison of techniques from place to place and from patient to patient is impossible without such information.

To be considered adequate in view of the foregoing objectives, the method of dosage calculation and presentation must satisfy certain criteria. These are:

1. Complete demonstration of the depth dose distribution throughout the entire irradiated volume of the patient so that, in particular, (a) the homogeneity of dosage within the tumor may be judged, and (b) unnecessary radiation is avoided outside the tumor bearing volume.
2. This presentation of data should be clear and simple to avoid misunderstanding and error.

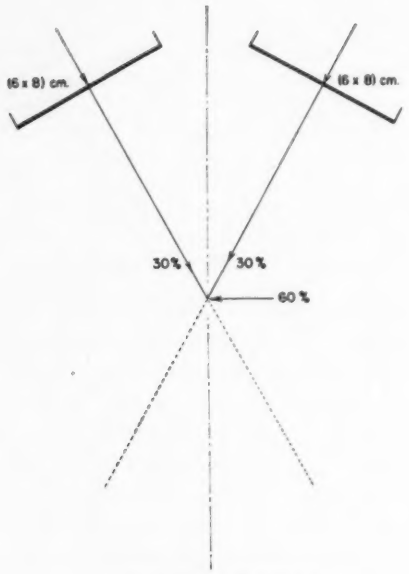
METHODS

Three general methods for determining tumor dosage and demonstrating the radiation distribution within the patient have been used:

1. Estimation from tables or curves of central axis depth dose (central axis estimation).
2. Point-by-point summation using isodose curves (point plot).
3. Direct isodose summation (direct composite plot).

1. *Central Axis Estimation.* The simplest procedure for obtaining some idea of radiation dosage in a tumor is the estimation of

* Presented at the Thirty-fifth Annual Meeting, American Radium Society, St. Louis, Mo., April 20–22, 1953.



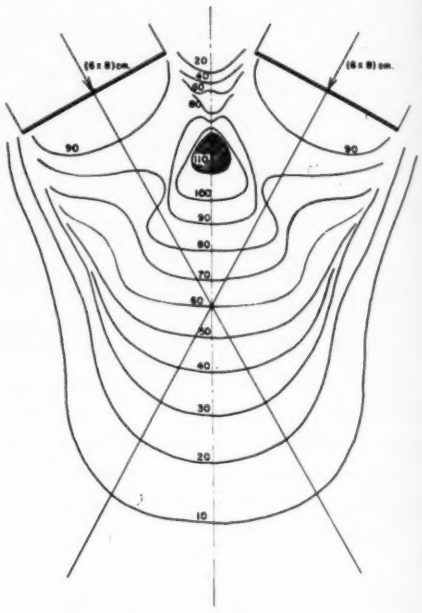


FIG. 1. Central axis dosage estimation is particularly inadequate in the case of oblique ports intersecting at fairly small angles. In this example, central axis dose estimation gives 60 per cent of the surface dose at the point of intersection. The maximum dose received in the tissues is nearly twice this, and is located much nearer the surface than the point of intersection.

tumor dose from central axis depth dose tables. There are several sets of tables available in which the dose on the central beam axis is expressed as a percentage of the dose incident upon the surface of the body. Perhaps the best known in this country are those of Quimby.[3] One of the earliest comprehensive sets of collected data due to Mayneord and Lamerton[5] has now been extended and brought up to date following a survey made by the Hospital Physicists' Association of Great Britain, under the editorship of Allsopp.[1]

The procedure for estimating tumor dose from these central axis tables is quite simple. The distance from the body surface to the tumor, or to the intersection of several beams in the tumor, is determined. The percentage contribution for each beam is read from the table, and the total contribution from all beams is summated. This summated figure is called "tumor depth dose." The method is quick, easy, and requires a minimum of apparatus and equipment.

This, however, is far from adequate in satisfying all the criteria for dosage calculation and presentation. The dose obtained applies to only one point within the patient. Central axis estimation provides no information about the homogeneity of the dosage throughout the tumor area, nor about the adequacy of coverage of the tumor volume by the beams of radiation. The therapist is not enlightened concerning dose distribution away from this point of beam intersection, and the location and intensity of high dose areas is not indicated.

A few illustrations will demonstrate the basic inadequacy of central axis estimation. In Figure 1, two oblique ports are arranged at such an angle and spacing that the dose at the intersection of their central axes is 60 per cent of that given to the surface. A complete isodose plot of this same arrangement made by one of the methods described below, shows that the actual maximum dose in the patient's tissues is 110 per cent, and that this maximum is placed

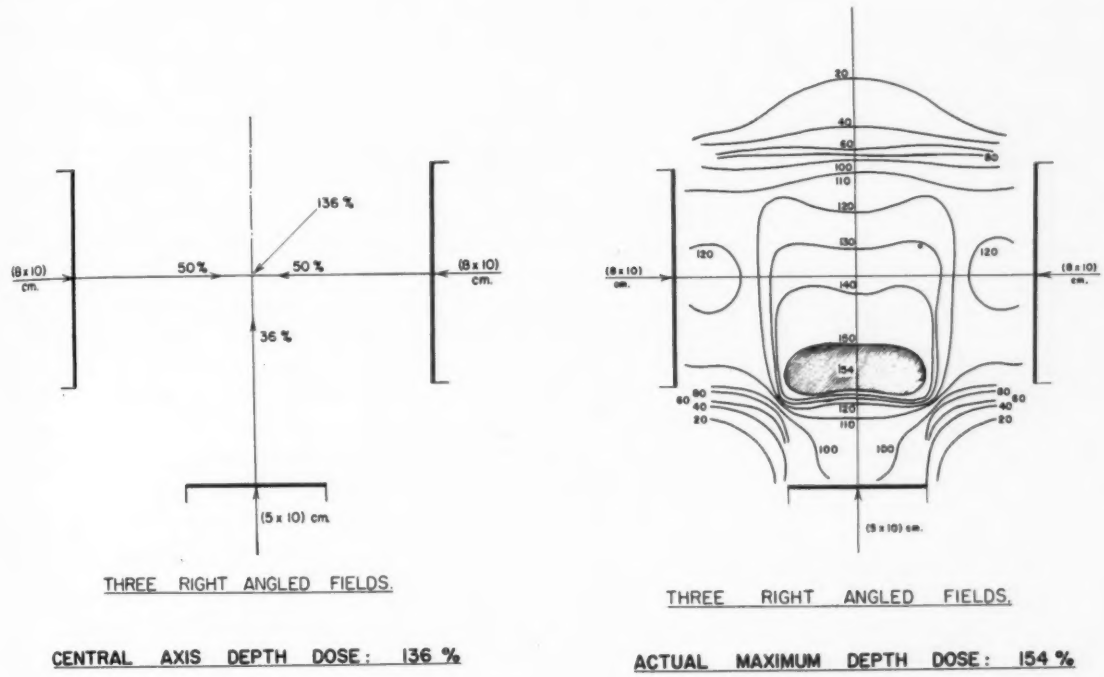


Fig. 2. If a third port is applied to one side of a system consisting of two coaxial opposed ports, the radiation intensity will be increased in the common field. Central axis estimation does not yield the location or intensity of the maximum dose.

considerably closer to the surface than the point at which the dose is estimated. There is no homogeneity of radiation field at the point of intersection, as it is located in a region of fairly steep field gradient.

Another example of error when using central axis estimation is shown in Figure 2. Here, a pair of opposing ports is used, with a third port at right angles to their common axis. The calculated dose at the point of intersection of the central axes is 136 per cent. The isodose plot shows the error in this estimation. The maximum dosage lies at some distance from the point of intersection and is approximately 20 per cent higher than that calculated.

2. *Point Plot Calculation.* A more complete representation of the radiation distribution in a treatment beam than that given by central axis tables, can be obtained by the use of isodose curves which give the complete depth dose distribution throughout a longitudinal section of the radiation beam. This representation showing the width and degree of coverage of the beam at any depth, as well as the radiation intensity within and outside the beam, obviously gives better insight into the total distribution of radiation than does a central axis depth dose table, and is to be preferred because of this.

The application of isodose curves to treatment planning is necessarily more complicated than is the use of central axis depth dose tables. The usual procedures involve the use of a full size, cross section outline of the patient at the level of treatment, to which the isodose curves are applied. The dosage is evaluated at various points within this outline, the number and location of these being dependent on the accuracy and completeness of dosage information required. These procedures are covered in detail by Paterson,[11] Lindsay,[8] Murphy and Reinhard,[10] and Johns,[7] and will only be outlined here.

In the application of isodose plotting either with a point plot or composite isodose summation, the first step is to construct a cross sectional outline of the patient at

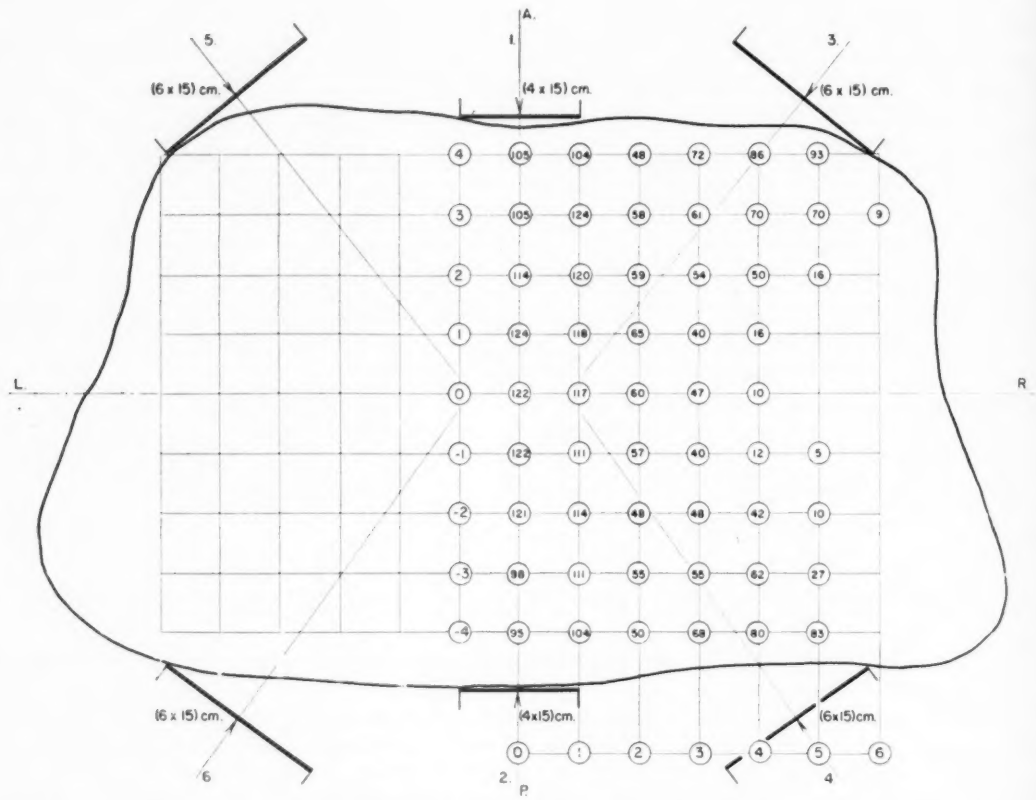


FIG. 3. Cross section of the thoracic region of a patient with six ports arranged to treat an esophageal lesion. A regular 2 cm. grid has been superimposed on the outline and the radiation dosage determined at the intersection of each grid line. Doses have been evaluated only on the right half of the outline as the dose distribution on the left will be a minor image of that on the right. This represents a "complete point plot."

the level of treatment.[9] This may be done in a variety of ways, one of the simplest being to use a lead strip or heavy wire that may be easily bent about the patient. If this is used, it is also necessary to measure the patient in treatment position to correct for possible deformation in removing the lead. Quick setting plaster bandage may also be used which, because of its rigidity, obviates the need for measurement. A device for obtaining body contours rapidly and which, it is claimed, is free from many of the disadvantages of the usual methods, has been described by Treherne and Greening.[14]

The outline is then transferred to a sheet of tracing paper or other transparent material, and the location of the tumor plotted in proper relationship to the surface contours. A number of points are then chosen where the dose is to be evaluated. Full scale isodose curves are applied to the outline in the positions that the ports are to be applied, and the contribution of each port to each point is tabulated (Fig. 3 and 4). After all the data are tabulated, the total dose at each point is summated.

This point plot method can be made as simple or complex as desired, and the amount of information obtained is directly proportional to the complexity of the method. If a small number of points is chosen, the procedure is quite simple and can be done in a few minutes. If more information is desired, the number of points at which the dose is evaluated must be increased. Figure 3 shows an arrangement in which the points are arranged in a regular 2 cm. grid. Since the port arrangement is symmetrical, the dose need only be plotted

over half of the area. This will permit adequate evaluation of dosage throughout the treated volume.

The amount of information given by the point plot procedure is much greater than can be obtained from central axis tables. However, the method leaves much to be desired in the satisfaction of the initial criteria. If a limited point plot is used in which only a few points are evaluated, the adequacy of the evaluation depends on the proper location of the points. If the points are selected so that they are representative of the high and low dose areas in the treated volume, the method is valid, and it is sufficiently simple to permit quick routine application. The proper selection of the points depends on prior knowledge of the approximate radiation distribution with the port arrangement used. Otherwise, the high and low dose regions in which the dosage must be assessed are not known, and the proper location of the points is uncertain. It is quite possible that areas of serious over- and underdosage may be missed. Also, a clear impression of the tissue volume raised to any particular dose is not given.

If the procedure is carried further, and a complete grid of points throughout the treated volume is evaluated, the information gained is increased, but the amount of work becomes vastly greater, as can be seen in Figure 4. The compilation and summation of the contribution of a large number of ports to a large number of points is a very slow tedious process. While a regular spacing of points at 2 cm. intervals gives sufficient coverage of the treated volume to eliminate the possibility of serious under- or overdosage, the data in the form of a number of isolated points are rather difficult to comprehend.

If complete isodose curves for the total of all ports are drawn by inspection superimposed on the 2 cm. grid, the presentation of data is greatly improved. Figure 5 shows the appearance of such composite curves. It will be seen that the complete radiation distribution is immediately and clearly visible. The areas of high and low

TABULATED DEPTH DOSES.

	0	1	2	3	4	5	6
4	93 7 5 105	90 6 8 104	48 48	72 72	86 86	93 93	90 90
3	74 6 5 5 5 7 105	75 8 30 4 5 124	5 48 5 58	61 61	70 70	70 70	9 9
2	57 13 16 8 12 8 114	58 11 30 9 5 7 120	5 42 5 7 59	49 5 54	50 50	16 16	
1	42 10 20 12 19 12 124	44 17 26 14 8 9 118	5 33 14 5 8 65	35 5 40	16 18		
0	30 28 27 25 17 15 122	32 23 21 18 12 11 117	5 24 21 5 5 60	23 19 5 47	5 5 10		
-1	22 18 13 18 13 18 122	22 33 19 25 10 7 111	5 16 29 7 57	5 30 5 40	12 12	5 5	
-2	15 52 10 18 10 17 121	16 46 10 29 8 5 114	10 38 5 48	43 5 48	42 42	10 10	
-3	11 69 5 5 5 5 98	11 60 5 30 5 111	5 45 5 55	55 55	62 62	27 27	
-4	8 87 96	8 85 11 104	50 50	68 68	80 80	85 85	

Fig. 4. Tabulated depth doses for the situation shown in Figure 3. The dose from each port to each point has been tabulated and summated. It is obvious from the complexity of this plot that this represents a tremendous amount of detailed work.

dosage are shown, as is the adequacy of coverage of the tumor bearing volume and the total volume irradiated to any given dose. Such a set of composite isodose curves more nearly satisfies the criteria than any of the other methods described above. Preparation of composite isodose curves by this method involves several steps.

1. Preparation of patient cross section outline.

2. Construction of a regular grid of points at 1–2 cm. intervals throughout the treated area.

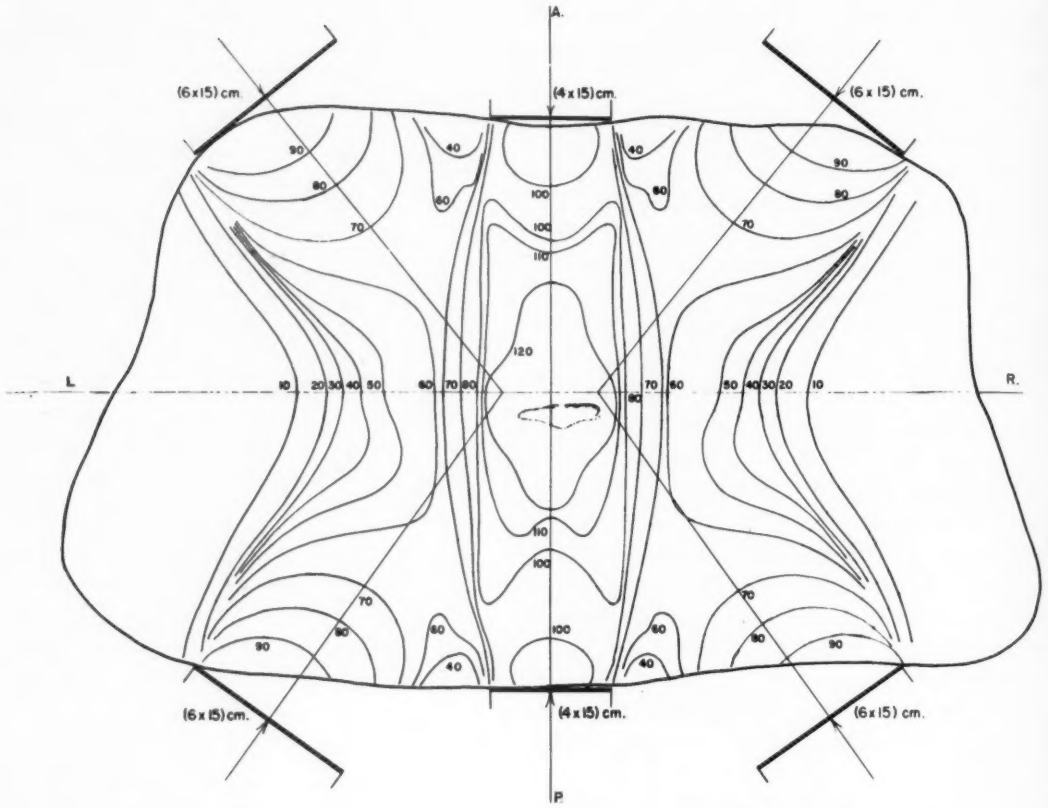


FIG. 5. In this figure, a complete isodose plot has been made for the treatment situation shown in Figure 3. This may be prepared from Figure 3 by inspection, or by the method of composite plotting to be described later. The superior clarity and completeness of this method of presentation is easily seen by comparison with Figure 3. (The small partly shaded area near the center represents the normal location of the esophagus.)

3. Tabulation and summation of the contribution of each port to each point (see Fig. 4).

4. Entry of the summated figures on the patient outline grid (Fig. 3).

5. Drawing composite curves by inspection from the grid.

Obviously this is a long tedious procedure, the longest part being the summation of dosage at each point. Generally this is too time consuming for routine application except with a large staff, as mentioned by Lindsay.[8]

3. *Direct Composite Plotting.* A much simpler and faster procedure for the preparation of complete isodose plots is the method of direct isodose summation or "composite plotting." This direct combination is a logical outgrowth of the use of isodose curves and, although widely used for many years, is not extensively discussed in published material on the subject; it is briefly treated by Johns.[7]

In essence, the procedure is very simple. It is carried out by superimposing and combining isodose curves in pairs until all of the desired fields have been included. This provides a direct picture of the complete distribution. The progressive development of the final plan is visible at each phase, and any required adjustment of port positions may be made at any time. With a relatively small amount of practice, the time consumed in preparing the average treatment plan is not excessive; for the same amount of information, it is much less than with the point plot method. This

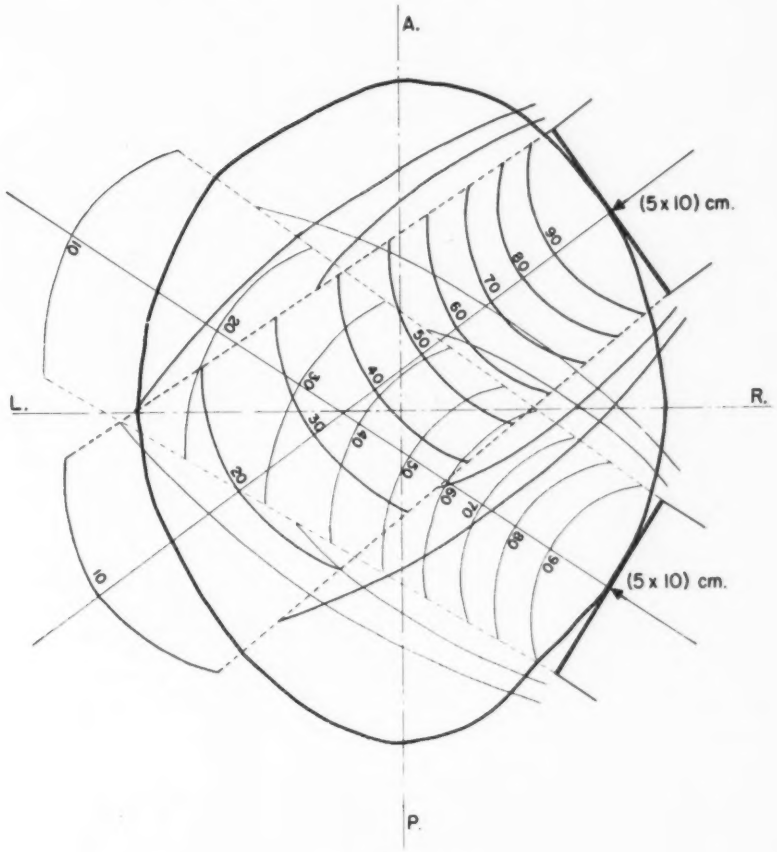


Fig. 6, 7, 8, and 9. These four figures show progressive steps in the preparation of a direct composite plot.
Fig. 6. In this drawing, two sets of isodose curves are shown as they appear superimposed on the outline of a patient's head.

procedure has been employed routinely in our department for some time. Residents with no previous experience soon acquire facility in its use.

In practice, a cross section outline of the patient with the position and extent of the area to be treated is drawn on tracing paper. Preliminary calculations, using central axis depth dose curves or tables determine the required number of ports to give the necessary depth dose. The isodose curves for appropriate port sizes, drawn in black on a translucent base, are then superimposed on the outline, and tentative positions and angulations are determined for the beams. It is convenient to do this and subsequent plotting with the sheets of tracing paper and the isodose curves placed on a viewing box with its face in a horizontal position. A sheet of tracing paper is then laid over one pair of isodose curves placed in the estimated positions. and points of intersection of the two sets of curves are then readily identified. Figure 6 shows the appearance of two superimposed sets of curves. After a group of points at the intersections of the two sets of curves which have the same value are identified, a smooth curve is drawn through this group of points on the overlaid sheet of tracing paper. The points indicated by circles on Figure 7 all will receive 70 per cent of the dose given to the surface. Those indicated by solid dots will receive 80 per cent, and so on. Figure 7

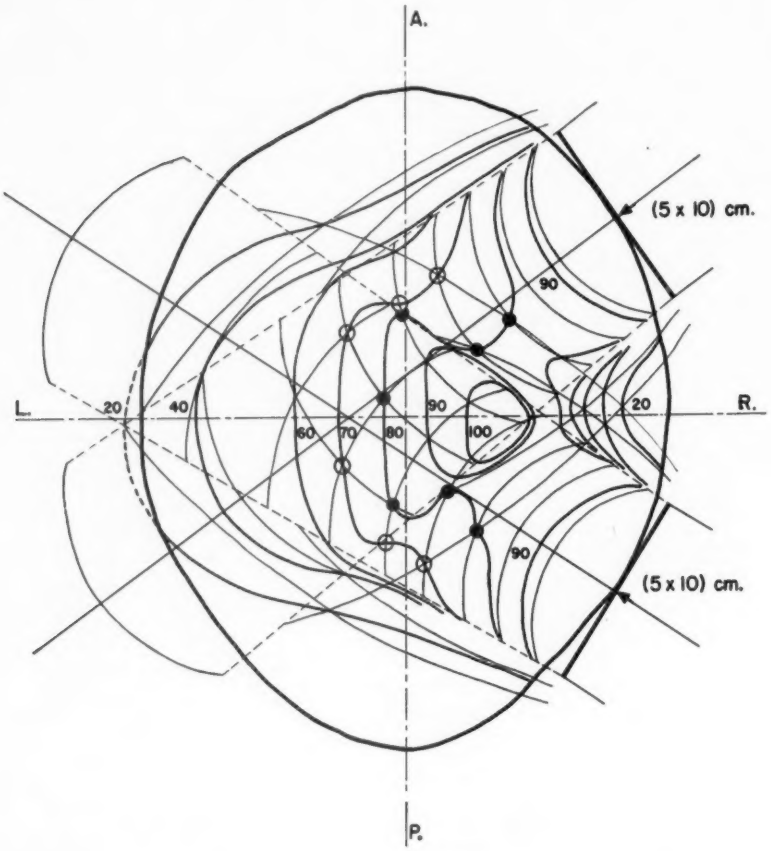


FIG. 7. Composite curves have been drawn through the intersections of the curves shown in Figure 6. Note that the solid dots are located at the 80 per cent intersections and the circles at the 70 per cent intersections. A smooth curve connecting each of the 80 per cent intersections defines the 80 per cent isodose surface in the plane of reference, the other composite curves are obtained in the same manner.

also shows a complete set of composite curves for this pair of ports as it would be seen on the light box. This composite curve represents the contribution of the right hand pair of ports in the final diagram. Figure 8 shows the composite summation of the left hand pair of ports which was obtained in exactly the same way. If now the right hand and the left hand composite curves are superimposed and combined, the final distribution seen in Figure 9 will be obtained. To avoid confusing detail, the isodose lines showing doses of less than 100 per cent have been omitted.

By adhering to a few rules for the direct summation of isodose curves, and with a little practice, the combining of these curves proceeds quite rapidly. These rules are:

1. Isodose curves of different value will never meet or cross in the composite plot.

2. Isodose curves are always continuous (forming closed loops), or end on the surface. There is an apparent discontinuity at the geometric edge of the beam; curves which run into this discontinuity will end on the surface at the beam edge.

3. When two curves from two different beams cross, they divide the field into four sections; of these, one will represent an area where the total dose is higher, and another, where the dose is lower than the sum of the two curves. As shown in Figure 10, the resultant composite curve passes through the other two sections.

4. The summation need only be carried out

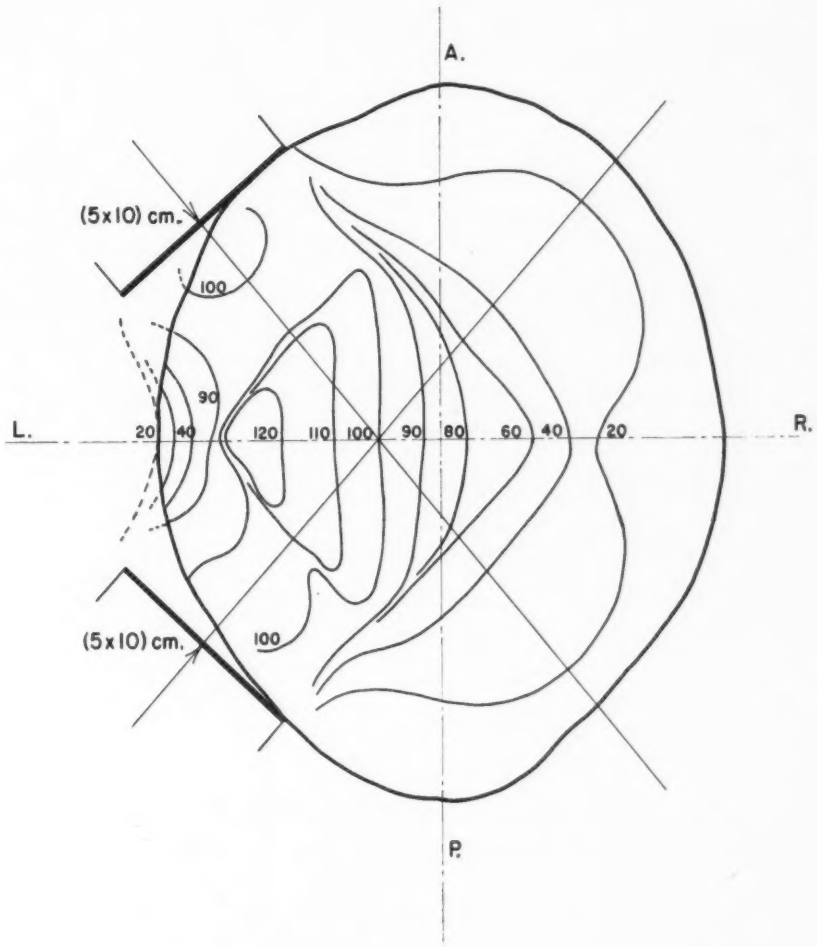


FIG. 8. Composite curves for a pair of ports on the opposite side of the head from those of Figure 6. The steps used in obtaining these are the same as in Figures 6 and 7, but have been omitted for clarity.

on one side of a plane of symmetry at any stage in the plan, as the second half of the distribution will then be a mirror image of the first.

5. Isodose curves are always perpendicular to such planes of symmetry.

The description of a method, where an attempt is made to cover all eventualities, always sounds much more complicated that the practical application actually is. When the first attempts are made to superimpose pairs of isodose curves and draw composite curves, the method seems forbiddingly complicated. With perseverance, however, the procedure rapidly becomes familiar, and after doing a few sets of curves, it becomes progressively easier. It is suggested that a start be made with opposing ports where the intersections are clear and the resultant curves intersect nearly at right angles. Then if successive plots are done with the ports intersecting at decreasing angles, it will be easier to follow the changes in shape and arrangement of the curves. After several composite plots have been done, one becomes familiar with the direction and course of the composite curves, and it is easy to place them in their proper relationship.

The preparation of a direct composite plot, as described above, gives maximum information with a minimum expenditure of time and effort. The actual time neces-

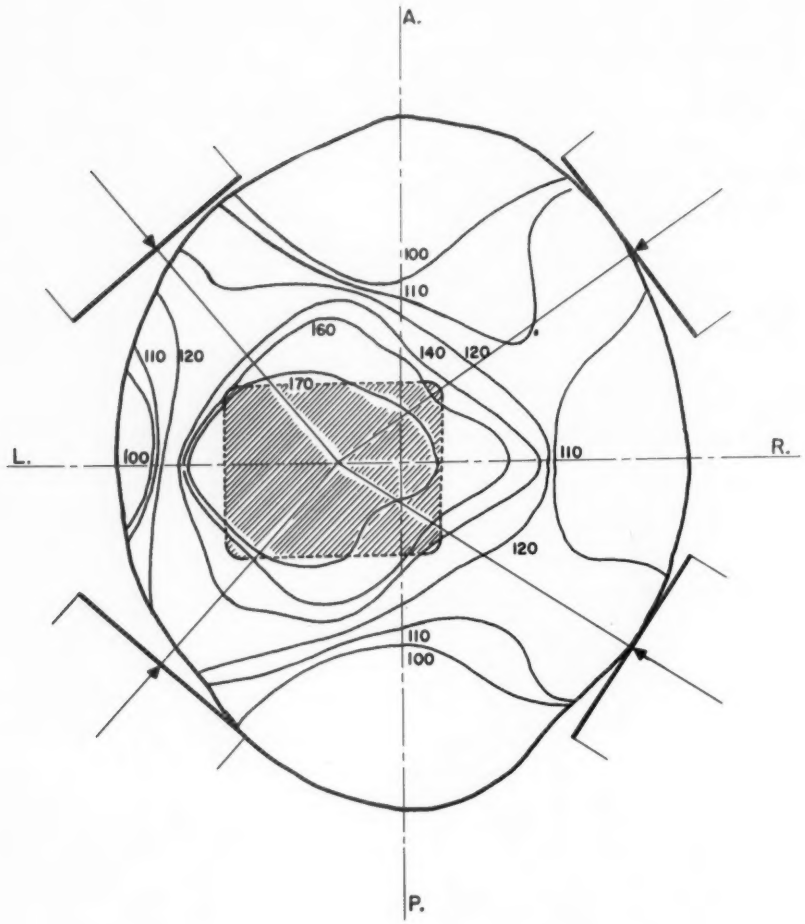


FIG. 9. Final composite curves for all four ports obtained by combining the composite curves of Figures 7 and 8. The cross hatched area indicates estimated tumor extent. Note that most of this tumor bearing area is contained within the 170 per cent curve and thus receives more than 170 per cent of the dose given to each port.

sary for the preparation of a direct composite plot is less than that required for the preparation of a detailed point plot in which the values only are tabulated. The entry of the values on the patient outline, and the drawing in of the isodose curves represent additional steps that greatly increase the expenditure of time and effort, when a point plot is used.

DISCUSSION

In intensive therapy where high doses are necessary which approach tissue and patient tolerance, central axis estimation does not give adequate information regarding dosage distribution. This may easily lead to excessive radiation in some areas, and inadequate irradiation in the tumor. In the palliative treatment of lesions which require low total dosage well below tolerance limits and where single ports or simple opposed port arrangements are used, central axis estimation may be adequate.

More complete information can be obtained with point plot methods using isodose curves, but the adequacy of the information depends on the number and location of the points chosen. If the points at

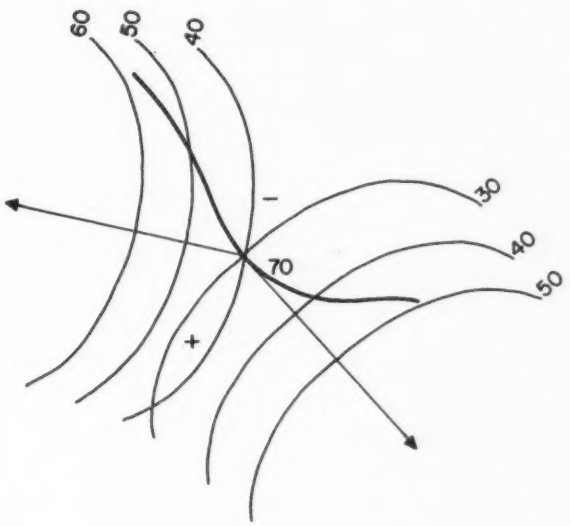


FIG. 10. Detail of small section of two superimposed isodose curves. In one sector (indicated by +) the dose is higher than at the intersection, while in the opposite sector (indicated by −) the dose is lower. The composite curve (indicated by the heavy line and the number 70) passes through the other two quadrants.

which dosage is determined are poorly located, the information obtained may be just as misleading as with central axis estimation. On the other hand, if a complete point plot is done, and composite isodose curves are drawn in by inspection, the final result can give a satisfactory degree of information.

Composite isodose plotting, as described above, will give a complete representation of radiation distribution within a patient more easily than with any other method. The extent of maximum dose is clearly shown, regions of excessive dose are clearly demonstrated, and the completeness of tumor coverage can be easily seen. The information is given in graphic form, and can be easily comprehended at a glance. A complete isodose plot must be considered the optimum presentation of dosage information.

In the use of any of these methods of dose computation and representation, it is necessary to remember that these show the radiation distribution in only one plane. The intensity of radiation outside of this plane of reference may vary appreciably from that shown. Figure 11 shows the magnitude of this effect in a symmetrical port arrangement using four ports. In the left half of this figure, a conventional distribution is shown; on the right, the distribution in a plane at right angles is demonstrated. This is drawn with nearly square ports. The use of more rectangular ports with their long axes at right angles to the plane of reference will help the situation at the expense of greater volume irradiated.

For either point plot or composite plotting with conventional isodose curves, it is also necessary that the central axes of all ports lie in the same plane. While methods have been developed which are suitable for the calculation of arrangements with

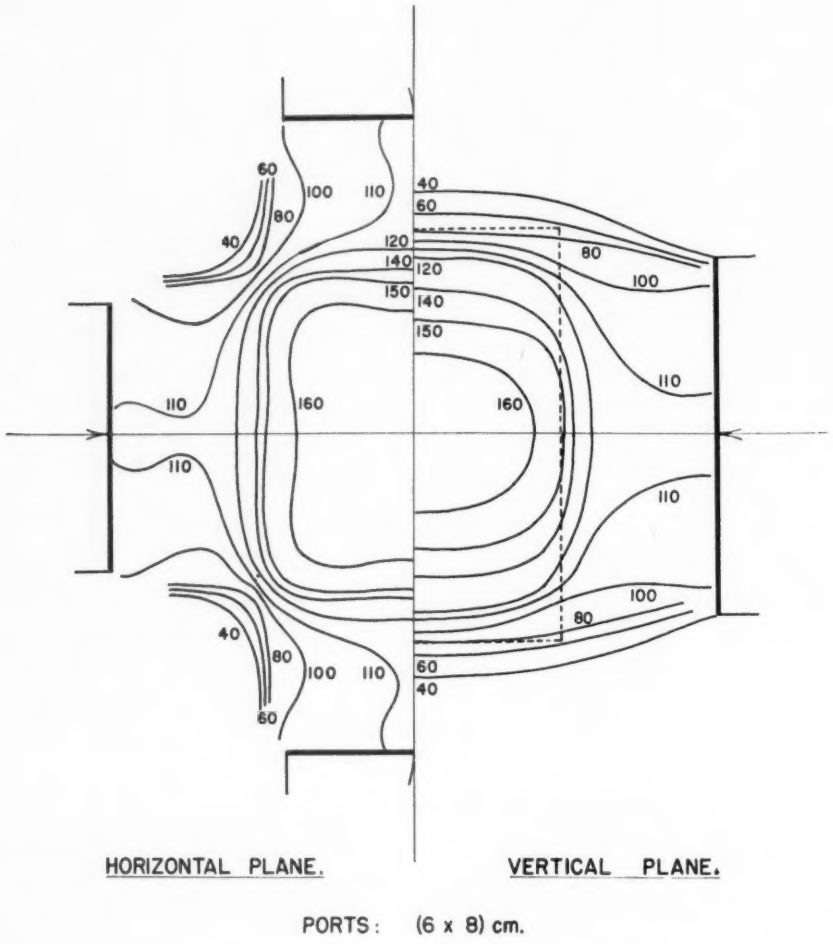


FIG. 11. Radiation distribution outside the common plane containing the port axes. On the left, half of a conventional representation of radiation distribution for four symmetrical ports in a transverse plane is shown. On the right, the radiation distribution in a longitudinal plane passing through one of the ports is shown. Note the limited spread of the radiation in this plane.

ports lying in several planes,[2,4,5] these require special equipment and add additional complication.

While these complete methods of plotting can be regarded as standard, they can be applied to every patient only in departments that have considerable personnel in proportion to the number of patients. Because of the man hours required, the application of these procedures may be impractical in the small department.

Various methods are available for simplification. One of these is to establish certain standard field arrangements and to precalculate composite plots for these treatment arrangements in patients of different sizes.[15] In this way, complete information can be obtained for each patient. Another procedure, when representative composite plots have been drawn for a particular field arrangement, is to use a limited point plot for each patient. The previous experience with composite plots will show the critical areas where dosage must be evaluated. A third situation exists in the palliative treatment for relief of pain where a simplified calculation may be quite satisfactory even though the total distribution

is not known. In such circumstances, limited point plot or central axis estimation may serve quite well.

CONCLUSION

In the preparation of a treatment plan for malignant disease, complete knowledge of radiation distribution throughout the irradiated volume is essential. Of the methods available for the determination of tumor dosage, central axis dose estimation is completely inadequate and may be dangerously misleading if it is relied upon for the evaluation of tumor dosage in all patients. Point plot determination of distribution is satisfactory if complete isodose contours are prepared, but this is a slow tedious procedure. Direct composite plotting gives a complete representation of radiation distribution with a reasonable expenditure of effort, and is the most satisfactory method available at the present time. Because of time limitations, it may not be possible to apply this procedure to treatment planning in every patient in a busy department. If complete composite plots have been precalculated for similar treatment plans, however, simpler methods of dose determination may be safely applied to the individual patient. Composite isodose curves represent a reasonably accurate demonstration of radiation dosage in one plane. However, radiation distribution in a patient is a three dimensional phenomenon, and further information would be desirable about radiation intensity outside the plane containing the central axis of the beams of radiation. The effects of bone and variation in soft tissue density also represent uncertainties which limit the accuracy of a conventional plot.[13] A more comprehensive procedure is needed to resolve these uncertainties, but at the present time, none is available.

SUMMARY

1. Criteria are stated for determining the adequacy of methods for tumor dose determinations.

2. Three methods for determining dose distribution are described and discussed.

3. The inadequacy of central axis tumor dose estimation is stressed.

4. Two more adequate methods for determining complete radiation distribution are described and discussed.

5. Suggestions are given for simplification of procedures in the small department.

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DISCUSSION

DR. JESSHILL LOVE, Louisville, Kentucky. There have been many occasions during the past few years that I have heard many of our colleagues and general practitioners of radiology say—"Case planning and cross section diagrams are important, but I do not have time for such things." It is my belief that if anyone attempts radiation therapy, he should avail himself of some method of dose estimation, dose calculation, treatment planning and dose distribution. Every patient who deserves treatment deserves some detailed attempt at dose planning and dose estimation. Malignant disease cannot be treated by a dosage table and the days of "hit or miss" therapy are past.

This paper today by Dr. Bloor has explained and clarified a method of dosimetry. It offers simpler and quicker plotting of the dose distribution; however, the point of overlap of two isodose curves often looks very confusing but attention and experience will speed the summation at various points. In some treatment centers this work is carried out by a computist. Every therapist, however, would benefit by manipulating his own isodose curves and planning various approaches and visualizing how it is possible to shape the contour of the summation isodose curve to fit the tumor area by moving the approach or weighting one of the beams on one side of the patient. It is important that one "sees or visualizes" the summation curves in three dimension volumes rather than in a plane. The value of estimating the tumor dose is appreciated when one evaluates what has been done at the end of the therapy schedule.

Patient management should consist of a careful history, staging of the clinical disease and planning the dosimetry to fit each individual case. The regimen, of course, can be changed at any time to fit the response of the disease while the patient is under treatment.

Such papers as the one today certainly help stimulate the interest in case planning to shape the course of treatment and management and then referring to the original plan at the end of therapy in order to actually determine the tumor dose delivered. This method is far more accurate than the common practice of directing the beam by guess and by sight and estimating the dose delivered by some "rule of thumb."

I would like to ask Dr. Bloor if he uses the same set of curves for various anatomical levels. Secondly, is absolute accuracy necessary? Third, it would be interesting if he would tell us the source from which he obtained his isodose curves.

DR. BLOOR (closing). I wish to thank Dr. Love for discussing this paper and for his comments. He has raised several questions which I will answer as best I can.

We do use the same curves for different anatomical levels. I presume that Dr. Love was referring to the fact that, at different levels in the body, there will be different degrees of bone absorption and air transmission as in the lungs. The presence of bone or air spaces will certainly modify the radiation distribution obtained from the standard curves. The amount of this modification is something that cannot be accurately determined on the basis of our present knowledge. However, to me, the important point to remember is that in treatment of the same body area in different patients, the same inconstancies in absorption will exist. Thus, while the values obtained from these calculations may not be true in an absolute sense, the inaccuracies will be similar in each patient, and the actual dosage calculated will be sufficiently accurate for comparative purposes. I believe that this partially answers the second question as well. While absolute accuracy would be desirable, and I feel that a great deal more information is needed regarding the effect of bone and air spaces on dose distribution, certainly a complete isodose plot based on the assumption of a homogeneous patient will give much more satisfactory information than can be obtained otherwise.

The third question concerned the source of our isodose curves. The curves that we are using were obtained from data which was very kindly furnished by Mr. Reinhard, the Radiation Physicist at Roswell Park Memorial Institute in Buffalo. Since the time that this paper was originally prepared, this information of Mr. Reinhard's has become generally available through the activity of E. Dale Trout of the General Electric X-Ray Corporation. For the first time in this country, isodose curves for a variety of filtrations, distances, and port sizes are now easily available to any radiologist, in this General Electric X-Ray Corporation set of curves.

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A STUDY OF RADIATION DOSIMETRY IN THE TREATMENT OF CERVICAL CANCER*†

By GEORGE C. LEWIS, Jr., M.D., RICHARD H. CHAMBERLAIN, M.D., JOHN HALE, M.S., *and* FRANKLIN L. PAYNE, M.D.

PHILADELPHIA, PENNSYLVANIA

REPORTED results indicate that the survival rates following radiation therapy for cervical cancer have undergone steady improvement.[2,5,9] At the Hospital of the University of Pennsylvania the apparent recovery rate has risen from 28 per cent to 46 per cent. The latter figure was attained at the cost of severe postirradiation complications in 14 per cent of the survivors.[4] These consisted of one death, several fistulas, bilateral ureteral occlusions, intestinal stenoses requiring resection or diversion operations, and massive tissue necrosis to be followed by complete vaginal closure. Careful study failed to explain these reactions because of inadequate data as to both the physical factors and the technical accuracy of the irradiation. The study did suggest indictment of the radium fraction. Of a group of patients who received about the same external irradiation the incidence of complications was more than doubled when the radium dosage reached or exceeded 5,000 milligram hours. Furthermore, through a failure to obtain roentgenograms of radium placements many malplacements were probably overlooked. One of the first roentgenographic studies (Fig. 1) demonstrated the potentialities for heavy damage to the rectum from marked displacement of the colpostat.

For these reasons a special program was instituted in an attempt to accurately evaluate radiation therapy in terms of tumor destruction and normal tissue response. This program includes careful pretherapy evaluation, controlled external irradiation, integrated radium source therapy and detailed long term follow-up study.

In the preliminary evaluation, the extent of the tumor is determined as accurately as possible. A complete study of the contiguous structures by endoscopic and roentgenographic means is conducted. Roentgen irradiation precedes radium therapy, and its dosage is governed by the physical qualities of the radiation, the factors of its application, the extent of tumor growth, and the anatomic dimensions of the patient. The primary aim is an even distribution within the parametrium of the maximum radiation that the patient's immediate local tissue reaction and general response will permit. At its completion the tissue dosage is estimated and the radium fraction is planned to integrate with this accomplishment. The achievement of radium dosimetry offered a major problem primarily because of the difficulties in accurately establishing the spatial relationship of radium sources and surrounding structures. The precision of these dosage calculations depends upon exact localization of both the elements and the pelvic viscera through visualization and careful measurement of their geometric relationships. Spatial distribution of radium sources has been studied previously, often without consideration of adjacent structures.[6,7,10] Orthographic pelvimetry, as described by Hodges and Nichols,[3] seems to offer promise in the calculation of complex dosage problems. Nichols and Carpender[8] have reported its successful application in the reconstruction of radium needle placements for cervical cancer.

Hodges and Nichols' equipment, consisting of a roentgenographic table and pantograph, was adapted to this study, with some changes in construction but without

* This project is supported in part by a research grant from the National Cancer Institute of the National Institutes of Health, United States Public Health Service, and in part by the Gynecean Institute of Gynecological Research of the University of Pennsylvania.

† Presented at the Thirty-fifth Annual Meeting, American Radium Society, St. Louis, Mo., April 20–22, 1953.

FIG. 1. Radium application (modified Paris) with colpostat displaced toward the rectum.

alteration of the basic physical principles. The roentgenographic table, end and side views of which are shown in Figures 2 and 3, is used for all roentgenograms during the study. The arrangement of the tube holders and cassettes for the production of anteroposterior and lateral exposures is demonstrated diagrammatically in Figure 4. The roentgen tube may be removed and accurately replaced by use of matched tapered pins and holes in cast aluminum mounts that are firmly attached to the table. The cassette holders and Bucky trays are mounted on the table at right angles to each other. The tube target distance is fixed at 36 inches. The centers of the beams which produce the exposures cross at right angles, and strike the films at exact right angles, at a point which is localized by lead markers imbedded in the table cover. Lines projected through the images of these lead guide marks cross at the point of intersection of the central beam and the film. Thus, the pelvis is converted into a geometric system in which the intersection of the central rays represents the zero point for x, y, and z axes. The horizontal central beam represents the x axis; the vertical central beam, the z axis; the y axis, not seen in this diagram, is horizontal and parallel to the planes of the anteroposterior and lateral roentgenograms. Due to the construction of the table the center of the horizontal beam (the x axis) is H cm. (14 cm.) from the anteroposterior roentgenogram. The center of the vertical beam (z axis) is V cm. (28.3 cm.) from the lateral roentgenogram. The y axis is H cm. (14 cm. from the anteroposterior roentgenogram and V cm. (28.3 cm.) from the lateral roentgenogram. The production of the roentgenograms can best be understood by a study of Figure 5. Lines *1* and *2* are in the same plane while line *3* is V cm. from the lateral film, and line *4* is H cm. from the anteroposterior films. The images must be reduced to their actual size before the original positions of the objects are reconstructed. This is done by utilizing Hodges and Nichols' pantograph which is illustrated in Figure 6. This produces drawings which represent the true dimension and relationship of the images that lie in two planes at right angles to each other. Thus, the proximity of the radium to any point within the pelvis may be calculated by reference to the geometric zero point and the x, y, and z axes. This reconstruction of the spatial relationship of objects permits the calculation of radiation dosages by use of the Quimby[1] tables for linear radium sources. By checking the accuracy of this technique in phantoms, it has been found that a maximum error of about 3 per cent is introduced into the final dosage calculations.

This physical accuracy does not alone insure success of the technique. Equally important is the visualization of all the vital pelvic structures. Major viscera which must be seen are the bladder, the lower ureters, the rectum, and rectosigmoid. Dosage calculations for the sigmoid are imprac-

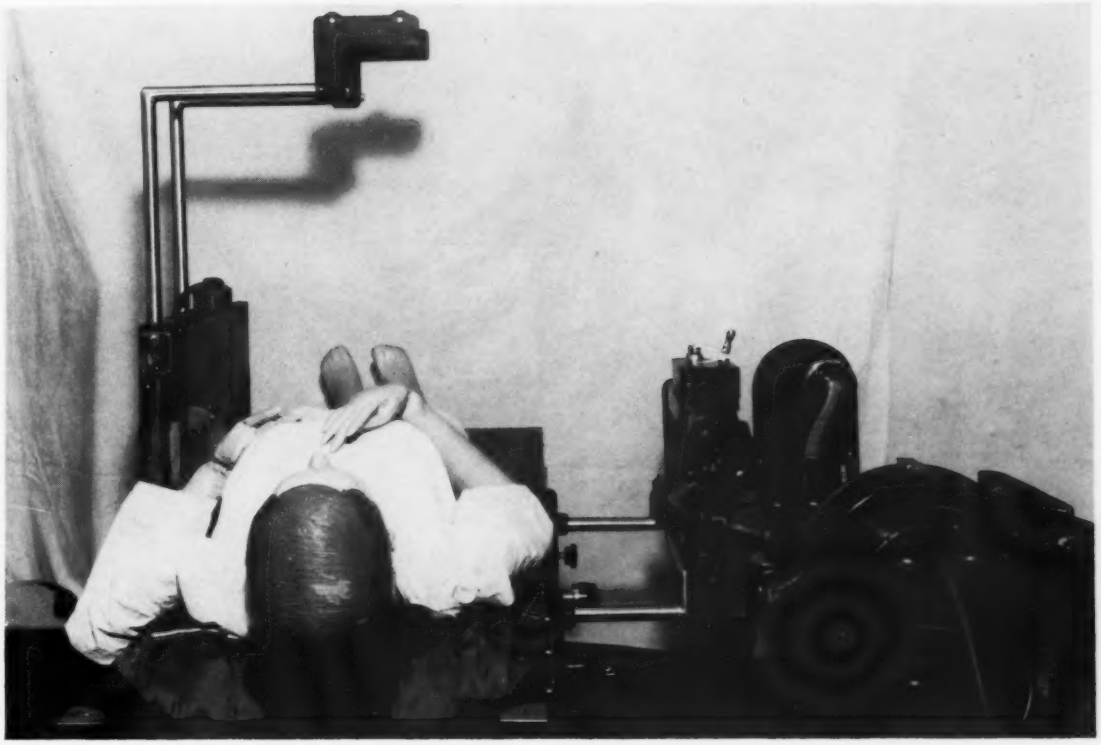

Fig. 2. Special roentgenographic table; end view.

Fig. 3. Special roentgenographic table; side view.

tical because of its motility.

The technique now utilized to visualize these structures is as follows: (1) Prior to radium placement, radiopaque catheters are placed in the ureters. (2) An individual-

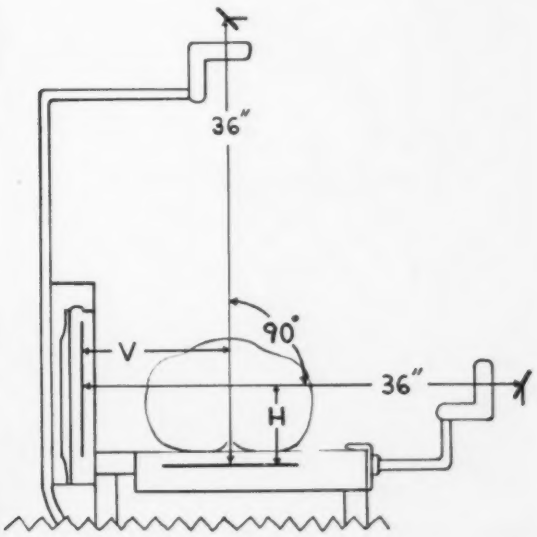

Fig. 4. Schematic cross section of roentgenographic table. The relationship of cassette holders and tube mounts is demonstrated.

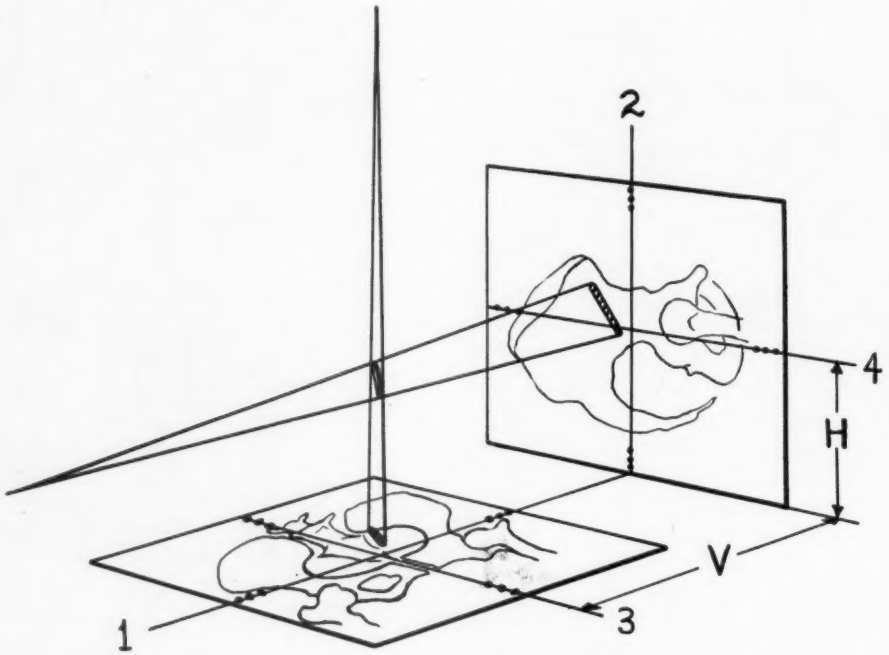

FIG. 5. Diagram illustrating the production of images by the orthographic method.

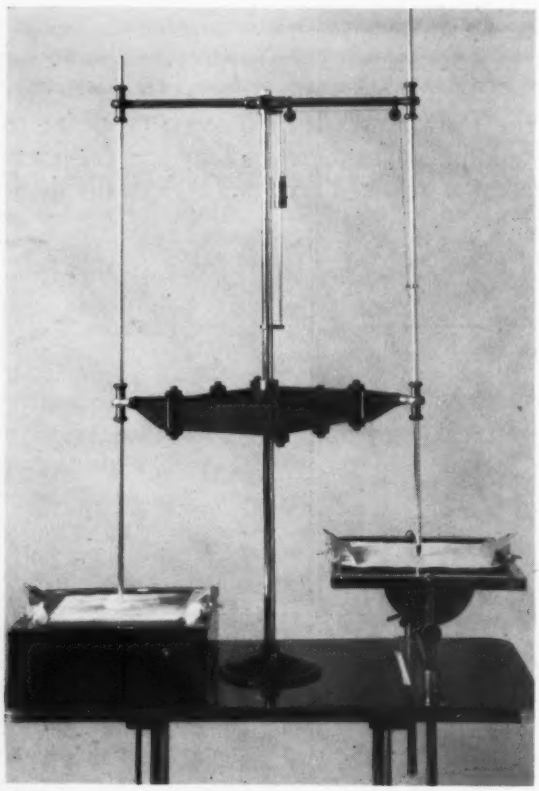

FIG. 6. Pantograph (Hodges and Nichols).

ized previously planned radium application is carried out under anesthesia. (3) A series of 10 by 12 inch roentgenograms are made using the orthographic technique: (a) (Fig. 7) an anteroposterior roentgenogram after filling the bladder with 50 cc. of 10 per cent neo-iopax;* (b) the bladder is emptied to permit clearer visualization of the ureteral catheters and a lateral exposure is completed (Fig. 8); (c) the bladder is then filled with 40 to 50 cc. of 25 per cent neo-iopax and the rectum with 150 cc. of 2.5 per cent suspension of barium sulfate (Fig. 9). A second lateral film is exposed.[9] The bladder is emptied and an anteroposterior roentgenogram is obtained to complete the series (Fig. 10). Prior to taking the last anteroposterior view, a lateral stereoscopic pair of roentgenograms is made without using the orthographic technique. Because the relation of the posterior wall of the bladder to the vaginal radium is obscure in the conventional exposures a special view is taken in the sitting

* Kindly supplied through the generosity of the Schering Company.

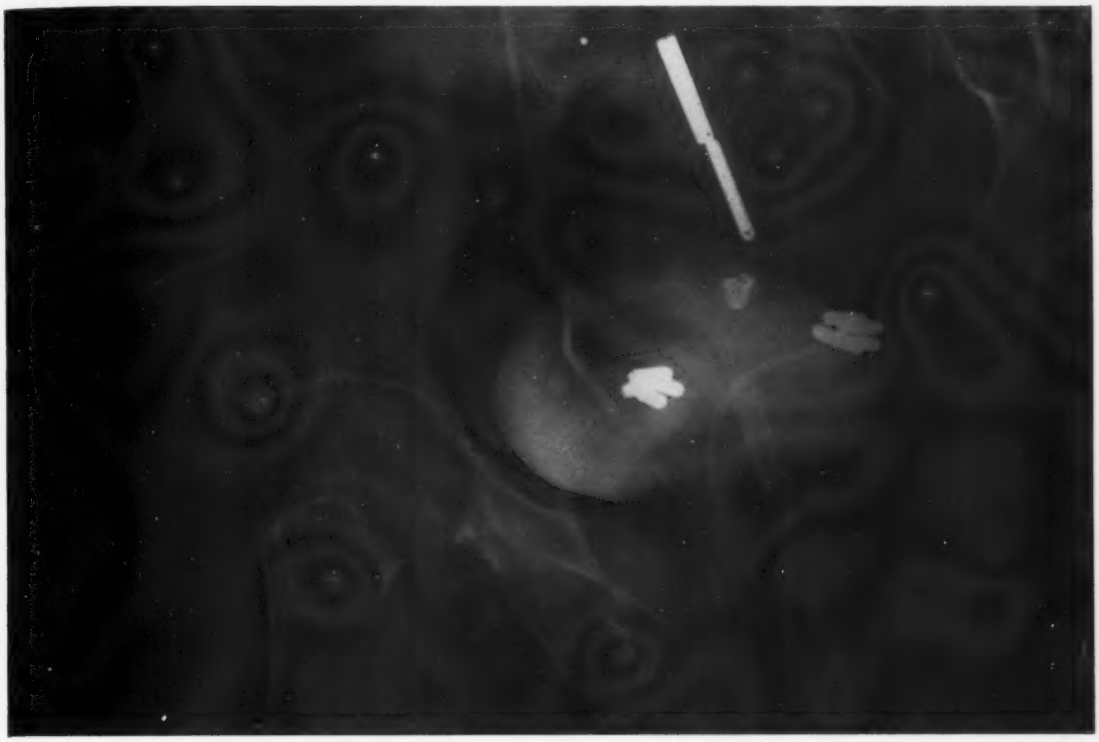

Fig. 7. Anteroposterior roentgenogram of the pelvis. The bladder contains neo-iopax. The course of the ureters is outlined by radiopaque catheters. A metal skin clip marks the cervix.

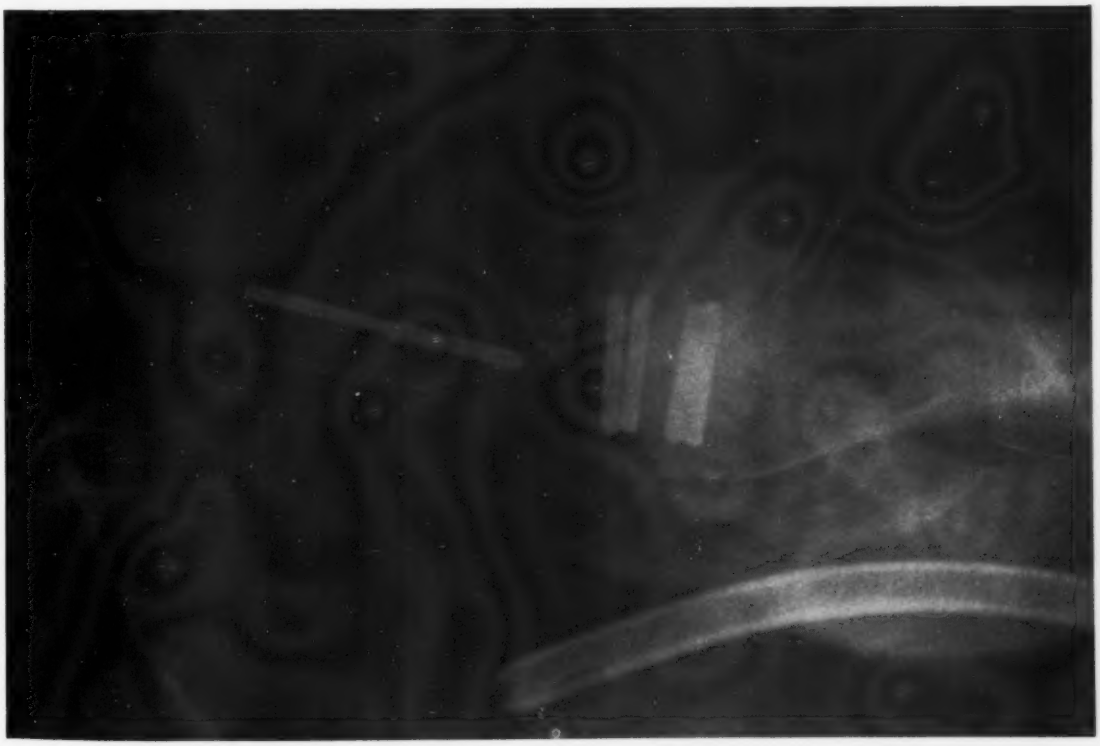

Fig. 8. Lateral roentgenogram of the pelvis. The bladder has been emptied to permit visualization of the ureteral catheters.

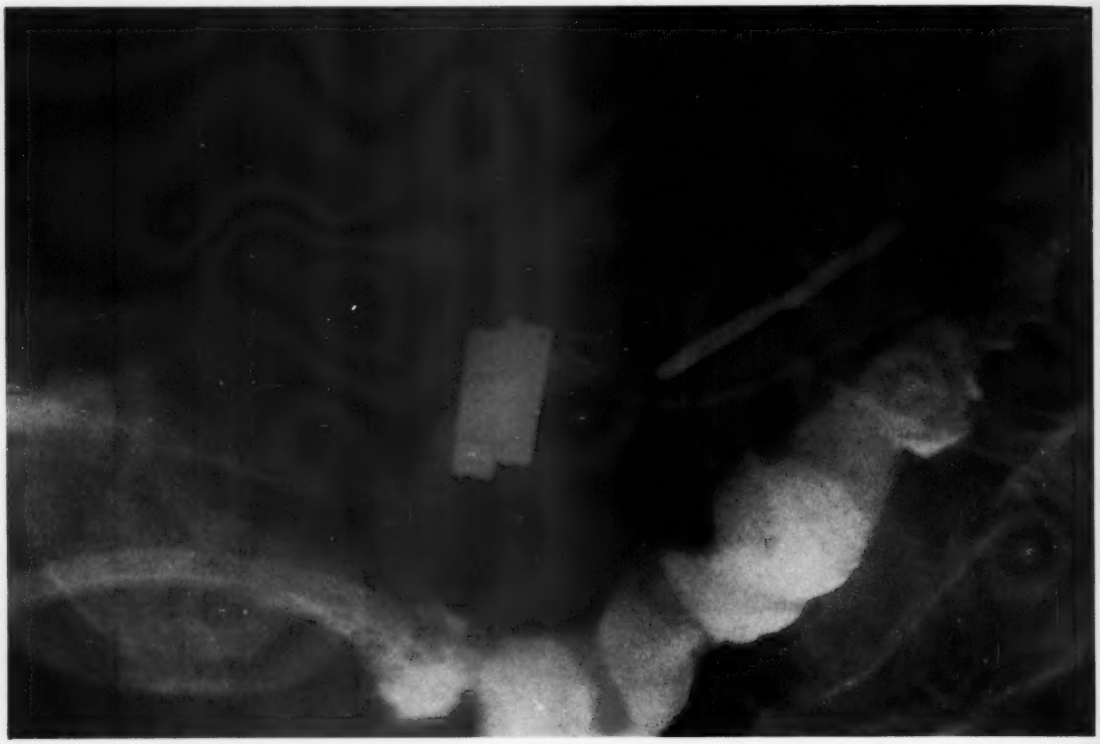

FIG. 9. The second lateral exposure is made with neo-iopax in the bladder and barium suspension in the colon.

FIG. 10. The final anteroposterior roentgenogram outlines the rectum and sigmoid colon. The neo-iopax is drained from the bladder prior to this exposure.

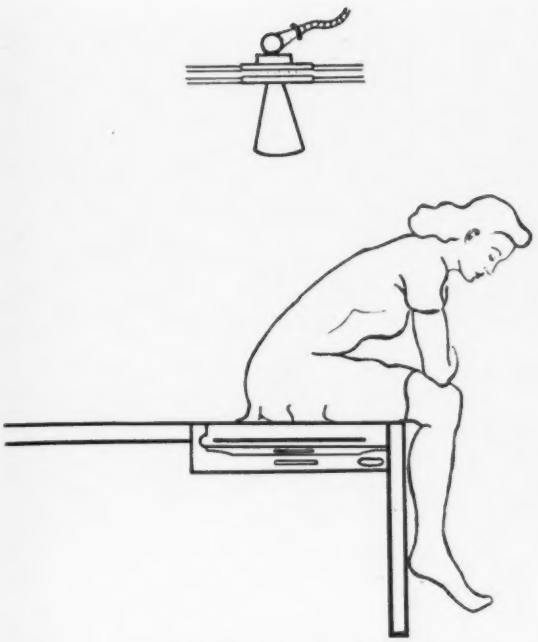

FIG. 11. Diagram illustrating the sitting position utilized to outline the posterior bladder surface. The greater trochanters are centered above the film.

position as shown by Figure 11. The rectum is lavaged free of barium and 50 cc. of 25 per cent neo-iopax is placed in the bladder before exposing the film. The resulting roentgenogram, shown in Figure 12, depicts a frequent finding: marked overlapping of the lateral margins of the bladder about the sides of the radium placed in lateral vaginal fornices. Calculations have shown that these lateral areas may be crucial in radiation therapy.

Although the technique of orthographic pelvimetry permits three dimensional isodose determinations, time limitations imposed by the calculations involved make it necessary at present to select certain points, zones, or volumes for therapy control. The radiation dosage is vitally important not only to actual and potential cancer bearing tissues but also to the contiguous structures. The need to consider both tumor destruction and preservation of normal tissue has been emphasized by Tod and Meredith[11] and by Tod.[12] Their points A

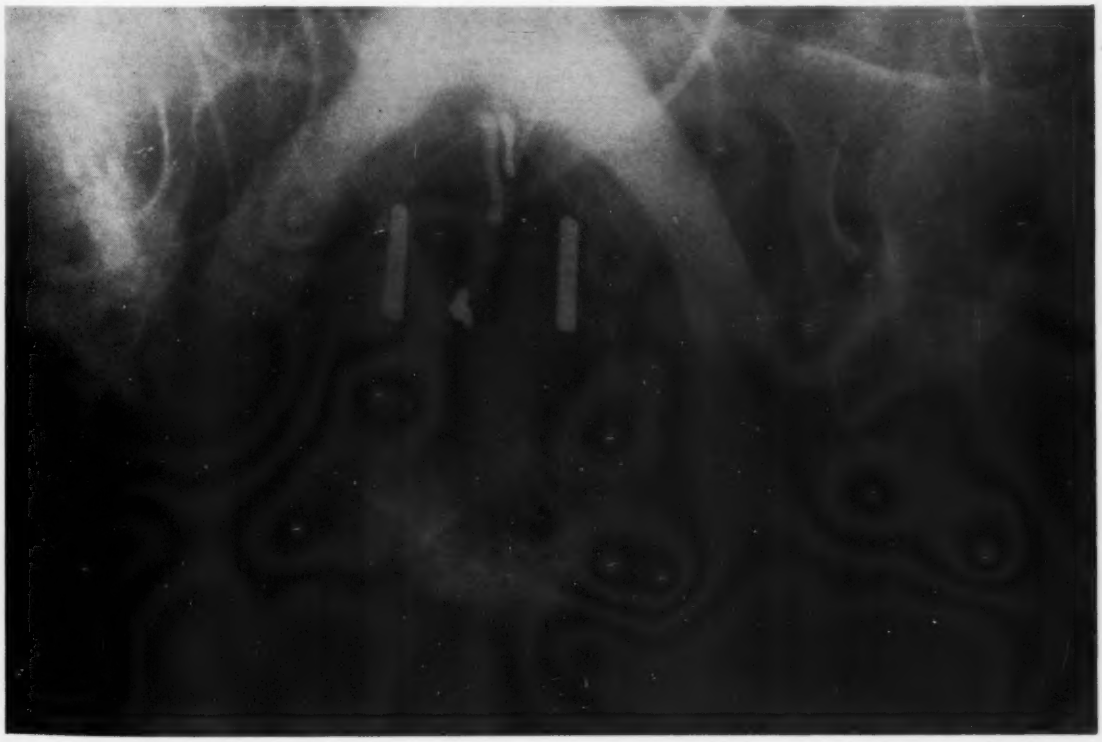

FIG. 12. The relationship of the vaginal radium sources to the bladder is shown by this roentgenogram made in the sitting position. Note the saddlebag appearance of the bladder.

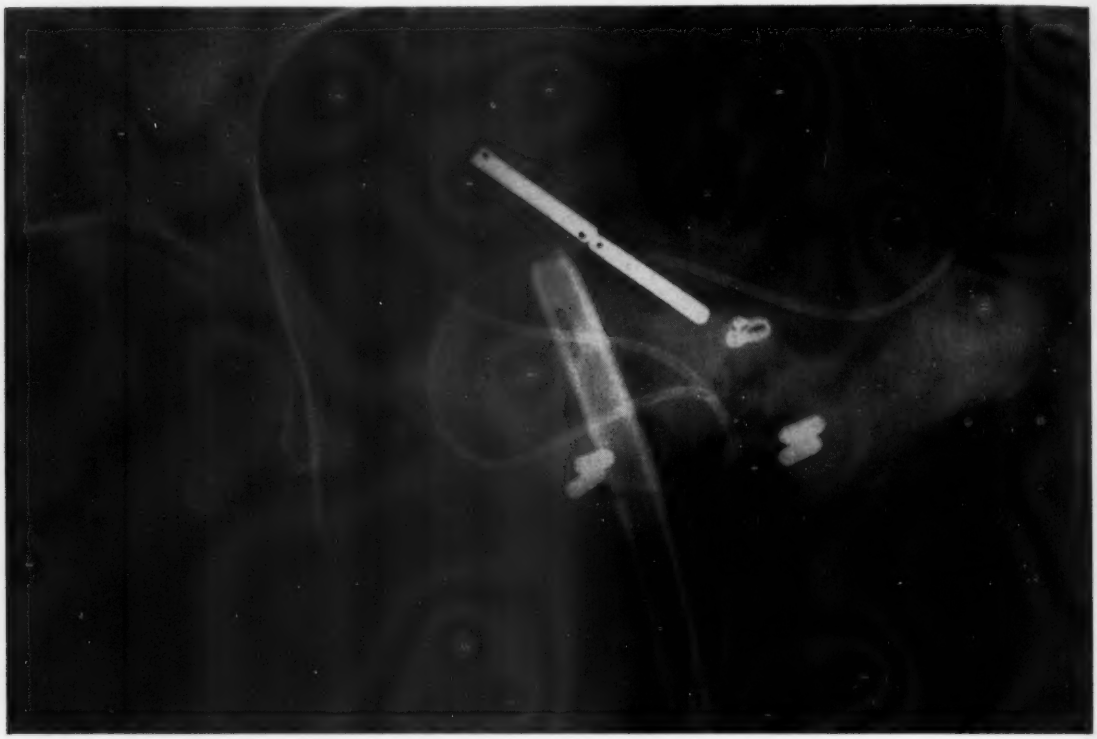

FIG. 13. Lateral displacement of the intrauterine tandem (modified Manchester technique).

and B appear most useful in defining tumor therapy when therapeutic data for different institutions are to be compared. While many difficulties arise in the clinical application of these points because of alteration of normal tissue dimensions by tumor, vaginal stenosis, or packing, they may still be considered valuable as physically defined but not anatomic locations. These limited points should not serve as the sole basis for the control of radiation that is spread throughout the pelvis. The technique described herewith permits the determination of dosage from radium sources to many critical points along the lower bowel, the bladder, and ureters, any one of which may be found to receive excessive irradiation. Thus, the sum of gamma and roentgen radiation to be delivered to these points must be balanced against that which is to be administered to the tumor areas. If the radium application causes an imbalance in the direction of normal tissue damage, revision of the planned therapy is indicated. Figure 13 illustrates such a case. Tumor bearing structures in the left side of the parametrium would have received inadequate radiation while the right ureter and right side of the bladder would have been overtreated, due to marked lateral displacement of the canal sources. The orthographic roentgenograms thus permit the therapist to visualize the malpositions that lead to defective radiation distribution and guide him in carrying out the proper corrective measures. Thus far, over half the radium applications have required some alteration to correct imbalances.

The use of the orthographic technique permits the integration of roentgen and gamma irradiation to form an accurate basis for correlation during the follow-up study. The final step in the program, the postirradiation evaluation, permits the detection of even minimal changes. For the first year the usual history and gynecologic examination is repeated at monthly intervals. Every three months, cystoscopic, urographic and proctoscopic examinations with barium enema visualization of the

colon are carried out even though the patient may be asymptomatic. During the later years intervals between the examinations are lengthened to six months. In this manner, the tissues to which calculated therapy has been administered are subjected to a thorough search for evidence of recurrent neoplasm or untoward reaction to radiation.

SUMMARY

At the Hospital of the University of Pennsylvania the five year survival rate of 46 per cent following radiation therapy for cervical carcinoma was accompanied by a serious complication rate of 14 per cent. Search for the faults proved fruitless because of the lack of information concerning tissue dosage both to tumor bearing areas and to contiguous structures. A program was instituted, by use of visualization, orthographic and pantographic techniques, to calculate depth dosage to both tumor and normal structures and to correlate this information with subsequent tumor destruction and normal tissue response in each patient. The mechanics of this program are described but the ultimate results, as to survival and complications, must await detailed long term follow-up study.

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DISCUSSION

Michel Ter-Pogossian, Ph.D., St. Louis, Missouri. I consider it a great privilege to be invited to discuss this paper presented by Dr. Payne. It is indeed a pleasure for me, a physicist, to discuss this excellent work presented by a physician, on a direct application of radiation physics to therapy. This situation exemplifies how fast this border between medicine and mathematical sciences fades away, or rather how broad becomes this overlapping area where physicians juggle with exponentials and where physicists do not mistake an obturator node for the component of a photographic shutter.

The project undertaken by Dr. Payne and his co-workers is of paramount importance in the radiation treatment of cancer of the cervix. This undertaking is a typical example of the modern tendency in this type of therapy, which is not any more the drive to deliver the highest dose of radiation to the largest volume of tissues but rather a concern over achieving this result with minimal damage to the, unfortunately, highly radiosensitive organs contained in the treated area.

The two tables presented by Dr. Payne at the beginning of his paper are a striking justification of the desirability of this project which he and his co-workers have undertaken. On the one hand, we have the five year survival rates obtained at the Hospital of the University of Pennsylvania; these results are excellent. I was particularly impressed by the very high percentage of survivals in Stages III and IV. On the other hand, we have the dear price paid for this 45 per cent survival rate, that is 50 per cent of significant complications. A correlation between these results and the amount of radiation delivered is definitely in order.

The first step described by Dr. Payne is an attempt to answer the important question of radium therapy of carcinoma of the cervix. Just how much radiation is delivered to various points within the pelvis?

Dr. Payne's solution of this problem is mathematical: It consists, first, in determining the exact relationship between the points of interest and the radium. Secondly, with the help of this information, in determining the dose of radiation received by these points.

Both of these steps should lead to the exact solution of the problem. The use of orthographic pelvimetry, as described by Hodges and Nichols for the determination of the relative position of the radium and various organs previously opacified by the use of contrast medium, is undoubtedly geometrically correct and leads to a good approximation of a three dimensional reconstruction of the pelvis containing the radium. Once the distances between the radium sources and the various points of interest are thus calculated, an accurate determination of the dose of radiation contributed by each one of the sources is easily computed through the use of Quimby's radium dosage table.

My only comment on this method is that of a lazy man. It sounds very time consuming. It seems to me that this dosage determination should be applied to every patient treated with radium. I know that in our institution this would not be practical because of the time involved. The question which comes next to my mind is: Could not the results sought by Dr. Payne be obtained with a lesser investment of time?

It seems to me that the direct measurement of these doses would lead to results at least as accurate as those obtained by three dimensional reconstruction. It is true that the lower bowel is hardly accessible to direct measurements. However, in most radium distributions the highest dose will be found in a very accessible region of the rectum; in addition, the position of the upper sigmoid is so variable that an accurate dosage measurement in this case is meaningless.

I should like to add to this discussion that I am particularly in agreement with Dr. Payne that all these radiation dosage measurements are of value only if the position of the radium remains reasonably constant with respect to the surrounding organs. Even a minor shift of the radium sources may create a high undesirable "hot spot."

I am sorry that the results of the measurements performed by Dr. Payne are not yet available. I hope that we shall see them in the not too distant future.

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ELEVATION OF CIRCULATING BLOOD HISTAMINE IN PATIENTS UNDERGOING DEEP ROENTGEN THERAPY*†

By E. C. LASSER, M.D.,‡ *and* K. W. STENSTROM, PH.D.
BUFFALO, NEW YORK AND MINNEAPOLIS, MINNESOTA

IN TWO previous papers,[7,8] the authors have indicated the relation between the absolute basal eosinophil count, adrenal cortical function, and the development of symptoms of radiation sickness in a homogeneous group of patients undergoing deep external radiation therapy for carcinoma of the cervix. Briefly, in an average thirty day treatment period, adrenal cortical function was found to be successively depressed up to a ten to fourteen day period at which time a gradual return to normal was begun. The basal absolute eosinophil count, and the symptom index (of radiation sickness) showed somewhat parallel changes, beginning to rise at about the tenth to fourteenth day of treatment and reaching a maximum at twenty to twenty-four days, followed by a return towards normal after this period.

It has been suspected, for many years, that radiation sickness was in some way linked with the liberation of tissue decomposition products acting as a general intoxicant. Ellinger[4] has pointed out, in the light of recent investigative knowledge, why histamine-like substances, or histamine itself, should be regarded as the toxic agent. Others have done likewise.[9] Segal[5] in several isolated determinations, has shown a rise in blood histamine values in some patients undergoing deep roentgen therapy.

The present study was undertaken to determine the relationship, if any, between the absolute basal eosinophil count, radiation sickness, and the circulating blood histamine.

METHODS

The absolute basal eosinophil counts and the symptom indexes were computed according to the methods outlined in the previous reports.[7,8] Twenty-one patients were included in the present study. As in the past, all determinations were carried out on patients receiving deep external roentgen therapy for carcinoma of the cervix. These patients constitute a somewhat homogeneous group for study, since there is a similarity in the quality, site, and schedule of irradiation they receive.

The basal histamine content of the blood was estimated according to Code's [2] modification of the method of Barsoum and Gaddum. Following extraction of the histamine component from a sample of whole blood, biological assay was carried out. This was done by matching contractions of guinea-pig terminal ileum suspended in Tyrode's solution upon addition of an unknown sample and upon addition of varying doses of a standard concentration of histamine. Contractions produced by the unknown blood samples falling between two known contraction waves 0.02 microgram per milliliter apart in value were estimated according to the heights of the respective contraction waves.

RESULTS

As in the previous reports, mean values for the three factors under study were computed for the entire group in successive five day intervals throughout the course of therapy. The number of determinations entering into each of these mean values and their numerical representations are produced in Chart 1. Graph 1 shows the graphic depiction of these values.

It will be noted that the absolute basal eosinophil curve and the symptom index curve show little variation over those previously reported.

As may be seen in Chart 1 and Graph 1, the histamine level is above normal range

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† Supported in part by the Atomic Energy Commission Contract AT-(11-1)-232.
‡ At present, Associate Chief Cancer Research Roentgenologist, Roswell Park Memorial Institute, Buffalo, New York.

DAYS	0-4	5-9	10-14	15-19	20-24	25-29	30-34	
Absolute Eosinophil Count	12	11	18	14	14	10	1	Number of Determinations
	133	*115*	*256*	*256*	*521*	*444*	*159*	*Average cells / cu. m.m.*
Histamine	8	7	13	11	12	11		Number of Determinations
	.122	*.105*	*.105*	*.102*	*.104*	*.109*		*Average value in gammas/ml.*
Symptoms	37	68	70	74	66	57	10	Number of Determinations
	30	*81*	*107*	*159*	*180*	*200*	*130*	*Average Symptom index*

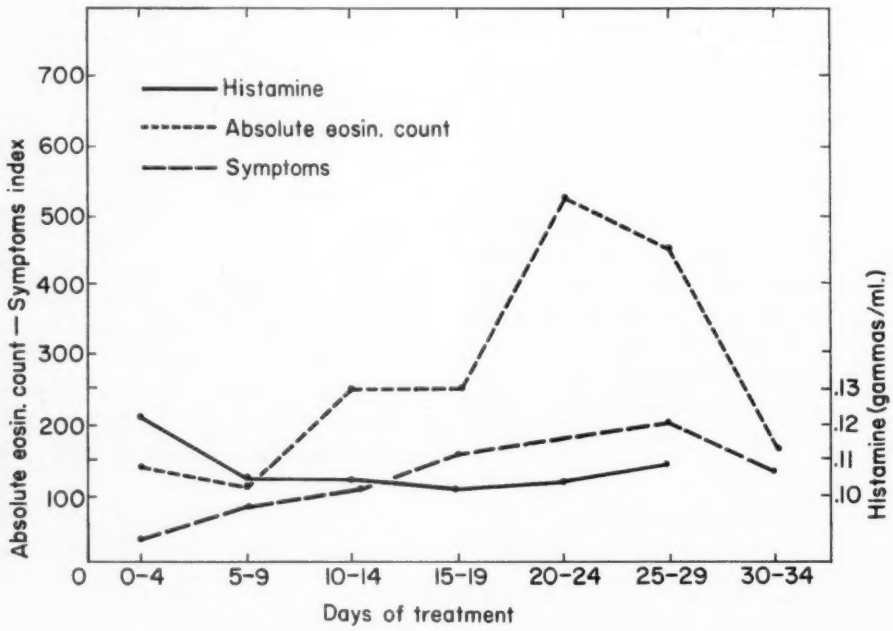
CHART I

throughout the treatment period. The level of circulating blood histamine is actually higher in the first four treatment days than in any other period of the therapy. After the initial drop there is relatively little change in the histamine level in spite of the gradual rise in the other two curves toward the end of therapy.

The normal blood histamine level is reported as varying from 0.01 microgram per milliliter to 0.08 microgram per milliliter and averaging about 0.04 microgram per milliliter.[6,11,12] Values above 0.10 microgram per milliliter are almost always regarded as abnormal.

COMMENT

Code,[3] in 1937, showed that in man, 70–100 per cent of the histamine extractable from whole blood was contained in the buffy coat layer of centrifuged unclotted blood. He concluded that the granular



GRAPH I

leukocytes are the main source of histamine in blood. Valentine, Pearce and Lawrence[14] found the average blood histamine value in chronic myelogenous leukemia to be 4.14 micrograms per milliliter. Randolph and Rackemann,[11] studying 9 patients with typical bronchial asthma, found the average histamine level at the height of attack to be 0.08 microgram per milliliter as compared to an average of 0.05 microgram per milliliter when free of attacks.

Our own study confirms the fact that elevated blood histamine levels are present in patients undergoing deep roentgen therapy. The slight fall in the histamine level in the five to nine day period may be related to the depression in the total granulocyte count encountered in these patients at about that period.

Other investigators have found that there is no constant relationship between the number of eosinophils in the peripheral blood and the levels of blood histamine.[11,14] This has likewise been true in our cases.

It is worth while noting that there were differences in the symptom curves and histamine levels when the group of patients under study were broken down into premenopausal and postmenopausal categories. As in the authors' previous studies,[7,8] the symptom index was considerably higher for the postmenopausal patients than for the premenopausal group and likewise the blood histamine level was slightly higher for each treatment period in the postmenopausal group than in the premenopausal patients. If an elevation of blood histamine is actually etiologically related to the production of symptoms in radiation sickness (not yet proved), it is interesting to speculate on the possible neutralizing effects of estrogen in the premenopausal group. Principe and Innocent,[10] for example, have shown that the addition of a water soluble synthetic estrogen (hydroestryl) abolishes the contractions of isolated guinea-pig intestine produced by histamine. Also Graham and Graham[5] have shown a diminished vulnerability of female Swiss mice to 400 r total body irradiation when given estrodial benzoate or estrone before exposure. Stilbestrol was inert in protective action in comparison to the natural estrogens.

CONCLUSION

1. Elevated blood histamine levels are present in a group of patients undergoing deep external roentgen therapy for carcinoma of the cervix.

2. The blood histamine level in these patients, while consistently elevated, does not appear to vary in proportion to the changes in the absolute eosinophil count or symptom index.

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Acknowledgment is made to Miss Virginia Betlach who carried out the histamine determinations.

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AN UNUSUAL RADIUM INCIDENT IN NOTTINGHAM

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THE INCIDENT

A UNIQUE and disturbing radium incident occurred early one Sunday morning in September, 1949, at the Nottingham General Hospital. A 25 milligram needle, after removal from a rubber vaginal ovoid, was placed along with some other needles in a small dish of water on a gas-ring to be cleaned by boiling. The nurse in charge was called away to attend to a patient, and the water in the dish was boiled "almost completely away." When the nurse returned, she removed the dish from the gas-ring, and placed the needles in a lead carrying block and then discovered that the 25 mg. needle was missing. The needle was eventually discovered on the floor and placed with the others in the lead block with only the eyelet protruding. It was some thirty hours after this incident, that is, on Monday morning, that the needle was sent to the Radium Department to be put away in the radium safe. Routine inspection of the needles at this time revealed that the conical point of the 25 mg. needle was missing. A series of radioautographs of the needle made on dental films suggested that most of the radium element had disappeared from the container. As the needle was known to be of the directly loaded type, there was no possibility of the radium still being intact inside gold cells. Further investigation led to the discovery that the radium salt was scattered about in a corridor and clinical room attached to the ward, where the needle had been boiled the previous day.

The corridor was a communicating bridge, built over a staircase, joining two rooms, one of which was the ward sisters' sitting room and the other of which was a clinical room. The gas-ring used to sterilize the radium needles was situated in the corridor (Fig. 1). The clinical room and ward had been used by a large number of people since the incident, so that the spread of radioactive contamination elsewhere in the hospital presented a grave problem.

DECONTAMINATION MEASURES

The first and most obvious measure undertaken was the evacuation from the grossly contaminated area of all personnel, including patients. Fortunately, this could be done with a minimum interference with the rest of the hospital, as the radiotherapy wards were in the old isolation block which was joined to the hospital by a single bridge built over the street. The windows in the clinical room were opened wide to achieve dispersal of radon. The next step was to prevent the spread of contamina-

Fig. 1. Corridor and window frames, partly demolished, where the incident occurred.

FIG. 2. The protective garments worn by workers during the decontamination work consisted of respirators, boiler suits, rubber boots and gloves.

tion, and clear up active material that had been carried about on shoes and clothing. To this end, several days were spent touring the hospital with a Geiger-Müller counter and collecting and condemning contaminated shoes, clothing, carpets, etc. Once all these articles were collected together, it was felt that there was little further danger of spreading radioactive material about the hospital and it was possible to return to the problem of cleaning the grossly contaminated area. The dangers of inhalation, ingestion and dissemination of active material from the area necessitated the workers wearing respirators (civilian duty type), boiler suits, rubber boots and gloves (Fig. 2). These protective garments were donned prior to entering the contaminated area and removed before leaving it. Only in this way could personnel be fully protected and the further spread of active material prevented. It says much for the success of these measures that during the lengthy process of cleaning the grossly contaminated area, the attempt to keep the contamination strictly localized was completely successful.

During the cleaning of the contaminated area, a strict inspection of personnel working in the region was maintained. Before going away from the site, all workers were monitored with a Geiger counter, special attention being given to finger nails, hair and shoes. In addition to this routine which was repeated several times daily, regular blood counts, breath tests and estimates of body content of radioactivity were arranged from time to time. One nurse who was being monitored with a counter against the chest to determine if any activity was present in the lungs, showed an alarming counting rate. Eventually, it was discovered that she was wearing a large luminous watch in her breast pocket. When this was removed, the counting rate dropped to normal background.

The clearance of the site necessitated the demolition of the small corridor where the incident had taken place. The staircase over which the corridor was built was boarded off to prevent the spread of contamination to lower floors and the rest of the department when the corridor was demolished. The work also required the removal of articles of furniture, sinks, floors and all strippable surfaces, such as paint on walls in the two rooms at either end of the corridor. Among the articles we had to dispose of were an easy-chair, a floor polisher, a table, trays, crockery, carpets, a wastepaper basket, a coal scuttle, a wash bowl, a kettle, curtains, blinds, weighing scales, books, etc. These were just a few of the articles we disposed of and give some idea of the scale of the destruction needed to clear away contamination.

When the grossly contaminated areas had been cleared, it was discovered that air borne contamination had spread through air vents into ceiling cavities. This arose from dispersal of active material in the early stages of the incident and during demolition work. The clearance of the ceiling

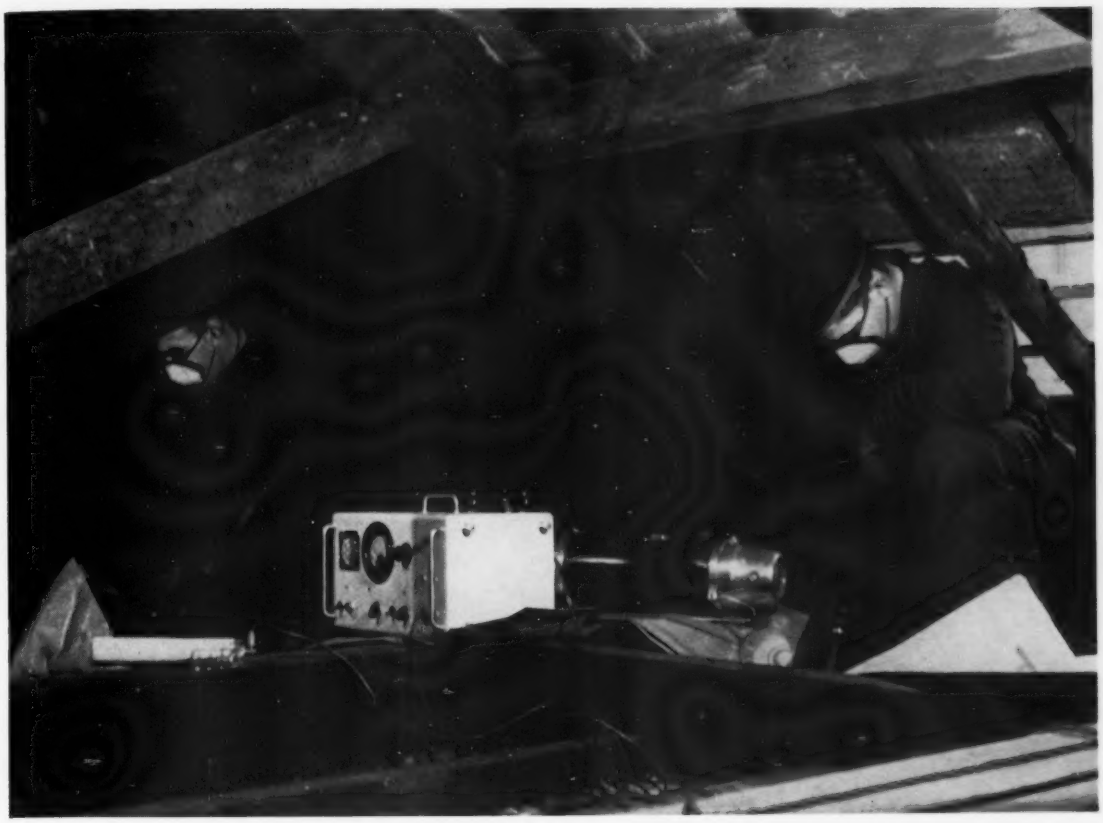

Fig. 3. Clearance of the ceiling cavities involved working with a Geiger counter to measure the level of activity as the contaminated rubble was removed. The figure also shows the vacuum cleaner that was used to collect active dust.

cavities necessitated the removal of a large quantity of building rubble left by workmen engaged on previous roof repairs, and added greatly to the physical labor involved (Fig. 3 and 4).

Grossly contaminated material was dumped in zinc troughs and the top concreted over. Less active material was placed in 1 cwt. sugar sacks composed of eight layers of paper (Fig. 5). The whole lot was placed in a courtyard outside the ward to await final disposal in some safe place. This proved to be not easy, as it was necessary to await agreement being reached between various interested Government departments. The first suggestion that the material should be dumped in the deep sea beyond the Continental Shelf was frowned on by the Ministry of Agriculture and Fisheries.

The contaminated material was finally disposed of by dumping down a disused mine shaft 900 yards deep. The shaft was situated well away from any used mine workings.

In attempting to restore the rooms to a condition where they could be used in safety in future, three hazards were borne in mind:

(1) External irradiation of the body by beta and gamma rays from residual radium.

(2) Internal irradiation from inhaled radioactive dust.

(3) Internal irradiation from inhalation of radon emitted from residual radium.

Accordingly, demolition was continued until tests indicated that the remaining activity was below 10 micrograms per room, which was regarded as permissible. As an additional precaution, the ceiling cavities

FIG. 4. General view of roof and scaffolding erected to lower contaminated material to the ground.

were sprayed with paint to seal any remaining active dust in position and prevent any further spread of air borne contamination. One of the most useful instruments in removing active dust was a vacuum cleaner which was used extensively during demolition and cleaning operations and subsequently dumped with the other active material down the mine shaft.

Several months elapsed before the residual activity was down to a satisfactory level and rebuilding could begin. It was difficult to estimate accurately the total amount of radium recovered. There was the uncertainty of such factors as deciding how much radon had been given off and how much could be assumed to be still occluded in the radium salt. Without an accurate assessment of the degree of equilibrium existing between radium and its disintegration products, quantitative measurements tend to be somewhat arbitrary. Also it was known that some radium salt went down the drains when the floor was washed just after the incident and before the discovery of loss was made. Since no activity was detected at the sewage disposal plant it was adjudged that the radium was caught in the drains. It was estimated that approximately 50 per cent of the radium had been recovered in the decontamination work and disposed of down the mine shaft.

After disposal of this material, final tests were made by the National Physical Laboratory to determine whether personnel had inhaled or ingested radium in quantities which might prove to be injurious. These tests indicated levels which, on the basis of existing knowledge about the effects of radium, should not lead to injury of any of the persons involved in the incident.

CONCLUSIONS

One of the most important issues arising from the incident is, of course, the precise reason why the radium container should have broken in the first place. The breakage took the form of an explosive rupture as a considerable quantity of radium salt was found deposited on ledges on a window frame several feet above the gas-ring on which the needle had been sterilized. To assume that the needle had been boiled dry and overheated to the extent that the gold solder joint had melted is an oversimplification of the facts. The other needles that were being boiled in the pot at the same time showed no signs of having been overheated. It is difficult to believe that one needle in a group can be so overheated as to explode and the other needles show no signs of damage at all. Any adequate explanation of this incident must take account of the following facts:

(1) The container was an old needle of the directly loaded type, and it may be that the radium salt was loaded in a damp condition. Gamma-ray bombardment may result in the dissociation of the water molecule producing hydrogen and oxygen, which in addition to helium forming from alpha particles, would tend to build up

Fig. 5. General view of zinc troughs, dust bins, and paper sacks awaiting disposal down the mine shaft. The troughs and dust bins containing grossly contaminated material were concreted over.

over a period of years a considerable gaseous pressure inside the container. A fairly slight overheating of the container could possibly cause sufficient increase of pressure to rupture the gold soldered joint.

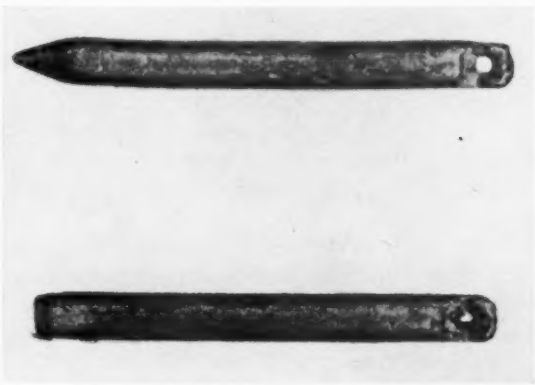

Fig. 6. Photograph (×3) of the ruptured needle alongside a needle of the same type and age. The break took place at the joint which was made on the tapered part of the conical point.

(2) The joint was not made on the barrel of the container as is the practice today, but was made on the tapered part of the conical point. This would tend to make a weak joint from a mechanical point of view (Fig. 6).

(3) The needle had been overheated by being allowed to boil dry in 1940. It is possible that this incident weakened the joint still further, though when the needle was submitted to the National Physical Laboratory, tests indicated that there was no leakage of radon through the joint.

(4) The needle, on being removed from the ovoid, was knocked against the side of the pot. This may have still further weakened the joint, so that on being placed in a pot of boiling water or on being boiled dry and overheated slightly, it may have been sufficient to cause the explosive rupture to take place.

Whatever view is taken of the precise

cause of this incident, the whole affair suggests that radium containers of this type and age are dangerous, and should be withdrawn from use.

Another conclusion which the incident suggests is that whatever method is used to sterilize radium needles, adequate safeguards should be taken to prevent the accidental overheating of radium containers.

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THE MECHANISM OF UROKON EXCRETION*†

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CLINICAL experience with urokon as an excretory urographic contrast medium has indicated that a higher percentage of satisfactory urograms may be obtained with this medium than was previously possible with other media.[1] In addition, opacification of the gallbladder has been noted during clinical urography and angiography with this substance. It seems desirable, therefore, to study the renal and extrarenal mechanisms involved in the excretion of urokon in order to explain these observations.

METHODS

Nineteen adult female mongrel dogs, previously perineotomized, and in apparent good health, were used in these studies, and all experiments were conducted on unanesthetized animals lying quietly on a table. These experiments can be divided into two general groups.

In the first group, studies were carried out to show the effect of urokon on the excretion of para-aminohippuric acid (hereafter referred to as PAH) and inulin, and the concomitant determination of plasma and urine urokon concentrations permitted determination of urokon clearances.

In twenty-one experiments urokon was administered by a single rapid intravenous injection similar to techniques used in clinical radiology. Inulin and PAH clearances were determined prior to and shortly after the intravenous injection of urokon, in eleven experiments. In twenty experiments urokon clearances were determined simultaneously with inulin clearances. It is realized that in the presence of rapidly changing plasma concentrations of urokon, renal clearance values are questionable, but our results in eighteen of these twenty experiments were so consistent that it was felt advisable to report these data. In seven experiments, inulin clearances were determined simultaneously with urokon clearances following subcutaneous administration of small quantities of urokon labeled with I^{131}.

In the second group of experiments, urokon labeled with I^{131} was injected intravenously. Urine was collected during the experiment, and at the end of an allotted experimental time the animals were sacrificed and the presence of urokon in various organs was determined by appropriate radioactive counting techniques. Clearances of both inulin and urokon were simultaneously determined in some of these animals prior to sacrifice and thus some animals contributed information to both groups of experiments.

Group I. Methods. Twenty-one experiments designed to study the effect of urokon on PAH and inulin excretion were carried out in the following manner. The animals were given water by gastric tube in amounts equivalent to 3 per cent of their body weight. An indwelling urethral catheter was inserted and urine was collected in graduated cylinders. This permitted measurement of urine flow at five minute intervals throughout the entire experimental period. After a diuresis of at least 0.5 ml. per minute was established, a priming dose of para-aminohippuric acid (0.02 gram/kilogram) and inulin (0.05 gm./kg.) was given intravenously. A sustaining dose of PAH (0.2 gm./kg.) and inulin (0.2 gm./kg.) was given subcutaneously in two divided doses about fifteen minutes apart. Three eleven to fourteen minute urine collections were made for PAH and inulin clearances and blood samples were taken at the beginning and end of each clearance period. At the completion of these clearance periods urokon was injected intravenously. Beginning five to fifteen minutes later, three more eleven to fourteen minute urine collections were made, and appropriately timed blood samples were obtained. Results of these experiments are reported as the average of these three periods. In some instances PAH and inulin clearances were determined twenty-four hours later.

* From the Mallinckrodt Institute of Radiology and the Department of Surgery, Washington University School of Medicine, St. Louis, Missouri. Read before the Association of University Radiologists, Chicago, Illinois, May 24, 1953.

† Aided in part by a grant-in-aid from the U. S. Public Health Service, No. A-96 (C) to Dr. C. Barber Mueller.

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In seven experiments, after an intravenous priming dose of inulin, a subcutaneous injection of inulin and urokon (30 mg./kg.) tagged with I^{131} was given in divided doses. This permitted the establishment of a constant, low plasma urokon concentration without the initial high concentration achieved after intravenous injection. Urokon and inulin clearances were determined simultaneously twenty minutes following the second subcutaneous administration.

PAH[6] and inulin[5] determinations were made on the trichloracetic acid filtrate of plasma.* Urokon analyses were performed by three methods, each of which gave satisfactory recoveries of known urokon samples. In the earliest experiments urokon was determined by the chemical analysis of its iodine content.† Subsequently, analyses were performed by either a spectrophotometric method using ultraviolet absorption at 238 mμ.‡ or by radioactive tracer techniques utilizing urokon labeled with I^{131}. With the latter method the radioactivity contained in 2 ml. of plasma, and in appropriately diluted urine samples was determined in a well-type scintillation counter with an estimated counting error of less than 3 per cent. All data are reported as milligrams of urokon, not urokon iodine (Fig. 1).

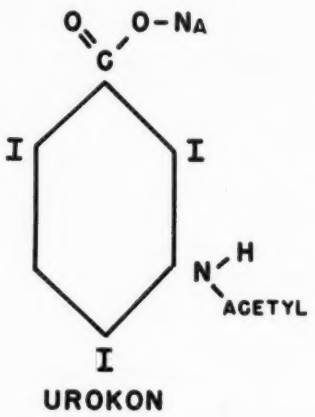

FIG. 1. Chemical structure of urokon (sodium, 2-4-6 triido, acetyl amino benzoate).

Group II. Methods. Eight experiments studying the distribution of injected urokon as a function of time were performed. Fasting animals were hydrated with water equivalent to 3 per cent of their body weight. After diuresis had been established and the bladder emptied, 0.5 gram of urokon/kilogram was injected intravenously. This urokon had been previously prepared by mixing carrier urokon with 1–2 mc. of urokon I^{131}. Urine was collected at half hour or hourly intervals. At the end of the experimental period, the animals were deeply anesthetized with nembutal, 50 ml. of blood was withdrawn by intracardiac puncture and the animals were then quickly eviscerated. After rinsing with water to remove excess blood, each organ with its contents was placed in a plastic bag. Thyroid, a portion of sartorious muscle and spinal fluid were obtained from some ani-

* Ten per cent trichloracetic acid precipitation of plasma proteins has been used with the diphenylamine analytical method of Rolf *et al.*[5] for the determination of inulin. The recovery of inulin added to plasma to achieve concentrations of 10, 15, and 20 mg. per cent has averaged 101 ±6.0 per cent when compared to a fructose standard and inulin in water solution.

† *Plasma Urokon Analysis by Iodine Analysis.* Deliver 10 ml. of trichloracetic filtrate of plasma into 125 ml. Erlenmeyer flask with ground glass neck. Add 15 ml. 4 per cent sodium hydroxide and follow with 15 ml. 6 per cent potassium permanganate. Attach to water cooled condenser and heat in steam bath for eighteen hours. After cooling add through the condenser 25 ml. water, 2 ml. 85 per cent phosphoric acid in 50 ml. beaker. Add excess 10 per cent sodium bisulfite and then 6 per cent potassium permanganate until light brown color appears, and immediately discharge this color with a few drops of sodium bisulfite. Adjust volume to 300–350 ml. with distilled water and the pH to 2.3 with phosphoric acid or sodium hydroxide. Add excess of saturated bromine water and boil for five minutes after the disappearance of the bromine color. Add a small crystal of salicylic acid and cool to room temperature. Add 1 ml. of starch solution and 4 ml. 10 per cent potassium iodide and titrate with 0.01 N sodium thiosulfate. Each milliliter of 0.01 N sodium thiosulfate is equivalent to 0.322 mg. of Urokon.

Urine Urokon Analysis by Iodine Analysis. Deliver 10 ml. urine to 500 ml. round bottom flask. Add 15 ml. 4 per cent sodium hydroxide and 75 ml. potassium permanganate (1:15). Attach to water cooled condenser and heat in steam bath for eighteen hours. Cool, add 25 ml. water, 10 ml. of dilute sulfuric acid (1–2 N) and follow with 50 ml. of water. Add 45 ml. of a 1:5 solution of sodium bisulfite; after cooling, add potassium permanganate until a yellow color appears, then add sodium bisulfite dropwise until the yellow disappears. Now add dropwise a dilute solution of potassium permanganate prepared by diluting 1 ml. of the 1:15 solution with 49 ml. of water, until a faint yellow appears. Add 1 ml. of starch solution and titrate with 0.02 N silver nitrate with vigorous agitation until the green precipitate formed just turns to canary yellow. Each milliliter of 0.02 N silver nitrate consumed is equivalent to 3.85 mg. of urokon.

‡ *Plasma and Urine Urokon Analysis by Spectrophotometry.* The absorption spectrum of urokon shows a maximum at wavelength of 238 mμ. In aqueous solutions Beer's Law is followed and concentrations are proportional to optical density. One milliliter of urine or plasma previously diluted with distilled water is placed in a 15 ml. centrifuge tube and to this is added 5 ml. of 0.5 N sodium hydroxide and 2 ml. of 10 per cent $ZnSO_4 \cdot 7H_2O$. The mixture is centrifuged and the supernatant fluid is read in a spectrophotometer at 238 mμ. against a distilled water blank. The absorption value of the supernatant fluid obtained from urokon-free plasma is subtracted from all plasma determinations. Urine blanks prepared in the same manner as the unknown, are determined and the absorption value likewise subtracted. The excretion of uriniferous substances having absorption properties at 238 mμ. depends upon time, and not upon urine volume, so aliquots used for urine blanks must represent dilution of urokon-free urine collected over a time period comparable to that of the unknown.

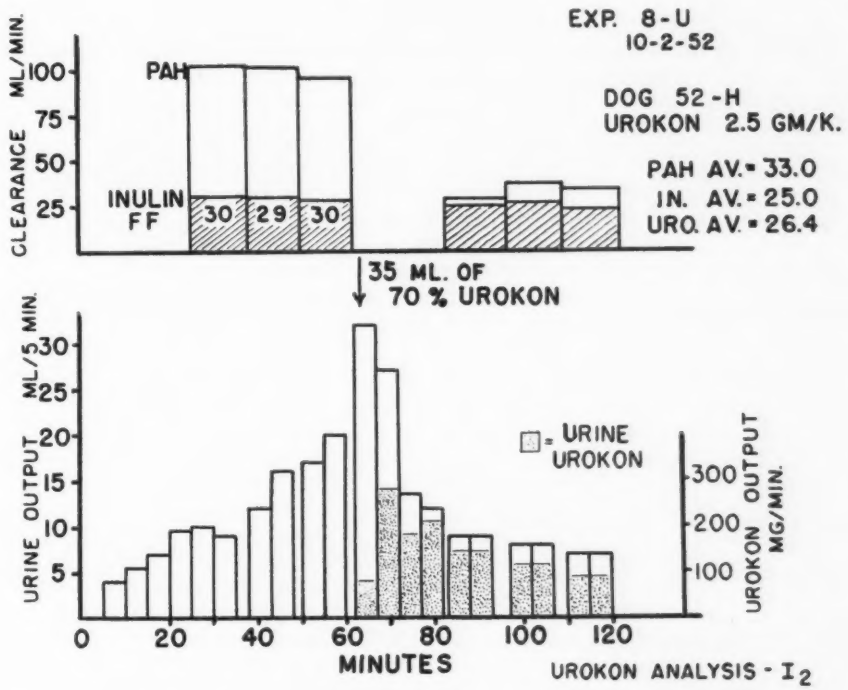

FIG. 2. Graphic representation of one experiment in which urine output and para-aminohippuric acid and inulin clearances were determined prior to and following the injection of 2.5 grams of urokon per kilogram. Following urokon injection, the PAH clearance fell from 100 ml./min. to 33.0 ml./min. The inulin clearance was unchanged at 25 ml./min. and the urokon clearance was 26.4 ml./min. Urokon analysis was carried out by the determination of its iodine concentration (see footnote †).

mals. The radioactivity contained in these specimens was measured with a scintillation counter at a distance sufficient to obviate errors due to differences in specimen size. Standardization was effected by appropriate dilution of aliquots of the injected radioactive compound. It was thus possible to calculate the proportion of the injected urokon remaining in each organ specimen. The data thus obtained are reported as percentage of the injected material.

RESULTS

1. *Toxicity.* Of 3 dogs given 5.0 grams urokon/kilogram (10 to 20 times the usual human dose), 2 died at two and eight hours respectively and 1 survived. The signs associated with the rapid administration of large quantities of urokon were hyperpnea, vomiting, defecation, convulsions and collapse. Autopsy on the 2 animals which died revealed a bloody peritoneal effusion in one, and no gross anatomical change in the other. Grossly and microscopically the kidneys of these 2 animals appeared normal. Renal biopsies taken twenty-four to forty-eight hours after the infusion of 2.5 gm./kg. in 3 animals were microscopically normal. All animals survived the administration of urokon in quantities of 2.5 gm./kg. or less without any apparent signs of toxicity.

2. *Effects of Urokon upon Inulin and PAH Clearances.* There was no change in the inulin clearance (glomerular filtration rate) following the intravenous injection of urokon in quantities of 2.5 grams or less per kilogram. In the 2 animals (57-H and 56-H) which failed to survive the injection of 5.0 grams of urokon/kilogram, there occurred a progressive decrease in inulin clearance. We feel that this is a manifestation of their pre-terminal collapse rather than primary renal damage. The animal which survived the injection of 5.0 grams of urokon/kilogram (Table 1, 52-H) showed

TABLE I

DATA SHOWING EFFECT OF THE INTRAVENOUS INJECTION OF UROKON UPON PARA-AMINOHIPPURIC ACID AND INULIN CLEARANCE

Dog No. Date	Urokon Injected gm./kg.	Pre-injection		Immediately Postinjection		24 hr. Postinjection	
		PAH ml./min.	Inulin ml./min.	PAH ml./min.	Inulin ml./min.	PAH ml./min.	Inulin ml./min.
52-H 9-9-52	5.0	115	28	28	29	111	30
57-H 9-16-52	5.0	192	46	26	25	Died in 2 hr.	
56-H 9-18-52	5.0	138	45	32	22	Died in 6 hr.	
60-H 9-30-52	2.5	129	38	37	28	116	31
52-H 10-2-52	2.5	133	44	49	41	133	33
60-H 10-9-52	2.5	128	36	42	33	—	—
52-H 3-19-53	0.25	121	34	88	32	—	—
61-H 4-18-53	0.25	169	65	109	57	—	—

no such fall in inulin clearance and it seems likely that urokon at this dose level has no direct toxic effect on the glomerulus.

The intravenous injection of urokon in quantities greater than 0.1 gm./kg. consistently produced a depression of the postinjection PAH clearance (Fig. 2). Postinjection PAH clearances in the 3 animals receiving 5.0 grams urokon/kilogram fell to values which equalled the postinjection inulin clearance. Following the injection of quantities less than 5.0 gm./kg., the depression of the PAH clearance was less marked and tended to vary directly with the amount of urokon injected (Table I).

The PAH clearance is generally considered to be a measurement of the effective renal plasma flow, and its excretion depends upon glomerular filtration and tubular secretion. Since the great depression of PAH clearances described above appears without concomitant decline in the glomerular filtration rate (inulin clearance), we feel that the reduction noted in these experiments represents a decrease in tubular excretory capacity for PAH and not a reduction in effective renal plasma flow.

3. *Route of Excretion of Urokon* (*Urokon Clearance*). To establish the route of urokon excretion, simultaneous inulin and urokon clearances were determined in nineteen separate experiments using 12 animals. Each experiment represents the average of three to five successive clearance periods.

In twelve experiments plasma urokon concentrations ranging between 20 and 1,000 mg. per cent were attained following single intravenous injections of urokon. In ten of these experiments the urokon and inulin clearance values were equal. (Urokon clearance values were significantly different from those of inulin in two experiments: in one it was 50 per cent higher and in the other 50 per cent lower. We are unable to explain the results of these two experiments.) The results of seven representative experiments are presented in Table II. Seven experiments were performed using urokon I^{131} given as a subcutaneous injection. This gave urokon plasma concentrations between 1 mg. per cent and 30 mg. per cent and avoided the high initial concentrations

TABLE II

INULIN AND UROKON CLEARANCES DETERMINED SIMULTANEOUSLY FOLLOWING THE INTRAVENOUS ADMINISTRATION OF UROKON

Dog No. Date	Urokon Injected gm./kg.	Range of Plasma Urokon during Clearance mg. per cent	Plasma Clearance		Remarks
			Inulin ml./min.	Urokon ml./min.	
60-H 10-9-52	2.5	530–350	33	32	Iodine analysis
52-H 1-1-53	2.5	1050–615	23	20	Analysis by U.V. absorption
52-H 1-1-53	2.5	455–390	23	20	Analysis by U.V. absorption
52-H 1-1-53	0.5	250–107	23	24	Analysis by U.V. absorption
70-H 2-2-53	0.5	163–70	45	40	Urokon I^{131}
61-H 1-24-53	0.1	30–13	49	50	Analysis by U.V. absorption
66-H 1-29-53	0.1	20–11	41	47	Analysis by U.V. absorption

previously achieved with intravenous injection. The data from these experiments are presented in Table III.

If one accepts the inulin clearance as a true measure of glomerular filtration, the amount of urokon excreted by the tubules may be determined by subtracting glomerular excretion (inulin clearance) from total urokon excretion. Thus, these experiments demonstrate that at low plasma levels a detectable, but small quantity of urokon is excreted by renal tubules in the dog. This quantity is of the order of 0.15 mg./min./kg. At plasma urokon levels of 30 mg. per

TABLE III

INULIN AND UROKON CLEARANCES DETERMINED SIMULTANEOUSLY FOLLOWING THE SUBCUTANEOUS ADMINISTRATION OF UROKON I^{131}

Dog No. Date	Injected Urokon gm./kg.	Range of Plasma Urokon Concentrations mg. per cent	Plasma Clearances		Tubular Secretion of Urokon mg./min./kg.
			Inulin ml./min.	Urokon ml./min.	
131-H 4-21-54	0.39	35–25	44	36	—
133-H 4-21-54	0.23	10–13	51	62	0.15
52-H 4-1-53	0.050	4–6	35	41	0.03
131-H 4-13-54	0.039	2.7–3.8	27	71	0.18
131-H 4-14-54	0.035	2.6–3.8	18	54	0.16
133-H 4-14-54	0.032	1.8–2.0	39	110	0.14
130-H 4-13-54	0.030	1.4–2.0	56	126	0.11

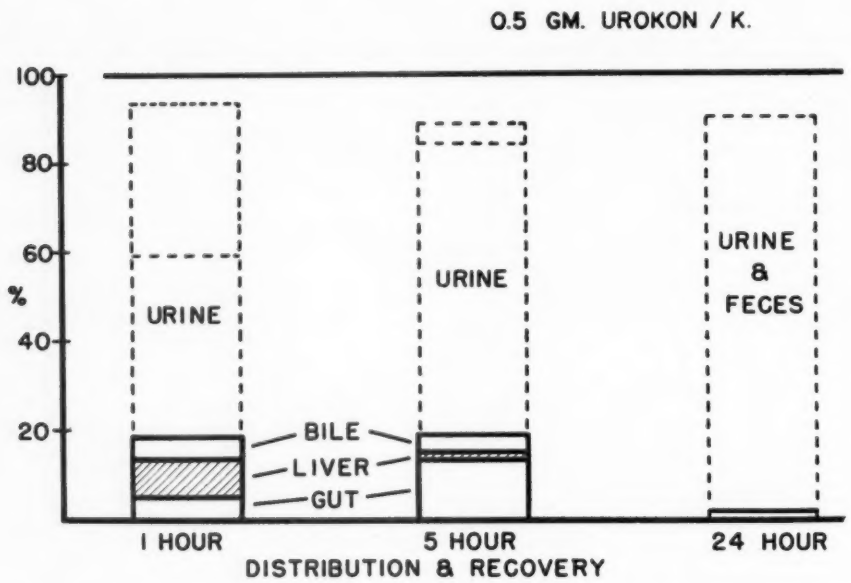

FIG. 3. Histograms showing the percentage of urokon found in the urine, bile, liver and intestine at one and five hours after intravenous injection. The open blocks at the upper end of these columns represent the urokon contained in all other viscera and that estimated in body fluids. The first and second columns represent the average of 3 animals. Only 1 animal was used for the twenty-four hour analysis.

cent or above, the tubular contribution represents a small fraction (less than 2 per cent) of the total urokon excreted. The glomerular filtration rate (inulin clearance) and the urokon clearance at high plasma levels are, therefore, essentially equal.

4. *Rate of Renal Excretion of Urokon.* The renal excretion of urokon in dogs receiving 0.5 gm. urokon/kilogram followed a constant pattern. Good agreement was obtained in nine experiments involving 5 dogs. The average urokon excretion was:

½ hour	25 per cent
1 hour	40 per cent
3 hours	57 per cent
5 hours	65 per cent
24 hours	90 per cent (1 dog)

5. *Distribution of Urokon in Organ Systems and Body Fluids.* The presence of a cholecystogram two hours following the intravenous injection of urokon drew attention to the fraction of the injected urokon that is excreted by the liver into the biliary tract and intestines. The experiments of Group II permitted an evaluation of the position of retained urokon in the various organ systems at one, three and five hours following intravenous injection. Examination of the data presented in Table IV indicates that approximately 20 per cent is contained in the hepato-biliary-enteric system. Most of the urokon which entered this system apparently did so during the first hour, while during the succeeding four hours a simple redistribution of urokon within the liver, gallbladder and intestine took place. The liver gradually excreted the urokon it had received and this urokon was subsequently found in the gallbladder and intestine. Although the total amount of urokon present in the gallbladder remained fairly constant during the first five hours after an injection, its concentration steadily increased and good cholecystograms were thus obtained (Fig. 3).

Almost all (90 per cent) of the injected urokon appeared in the urine and feces within twenty-four hours. The urinary excretion must account for the major portion excreted in twenty-four hours since the intestine and gallbladder together with their contents contained only 0.7 per cent of the injected urokon at this time. It therefore seems likely that urokon is re-absorbed

TABLE IV

TABLE SHOWING THE PROPORTION OF INJECTED UROKON PRESENT IN VARIOUS ORGAN SYSTEMS FOLLOWING RAPID INTRAVENOUS INJECTION OF UROKON. THE QUANTITY OF UROKON FOUND IN EACH ORGAN REPRESENTS THE ACTUAL ANALYSIS OF UROKON I^{131} WHILE THAT FOR THE BODY FLUIDS IS DATA DERIVED FROM RADIOACTIVITY CONTAINED IN SAMPLES OF PLASMA, WHOLE BLOOD AND MUSCLE

Sacrificed at	1 hr.		3 hr.		5 hr.		24 hr.	
Dog No. and Date	69-H 12-17-52	70-H 2-2-53	71-H 2-2-53	62-H 12-17-52	68-H 2-17-53	75-H 2-17-53	74-H 2-17-53	76-H 2-5-53
Injected amount gm./kg.	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5
Total gm.	5.0	3.2	4.9	3.1	4.6	4.5	3.6	2.0
Recovered amt. per cent								
Urine	46.0	41.0	41.0	52.1	63.0	67.0	67.0	89.0
Liver	2.07	9.2	6.5	0.9	0.6	0.96	0.5	trace
Bile	1.64	4.5	4.9	0.8	2.4	4.8	2.4	trace
Intestine (stomach, small intestine, colon)	2.16	5.2	5.2	7.9	4.0	13.1	14.1	trace
Kidney, Pancreas, Spleen, Lungs	1.2	2.1	2.3	1.3	0.58	1.1	1.1	trace
Plasma		7.2	5.9			0.9	0.8	trace
Red blood cells		1.3	1.2			0.56	0.5	trace
Interstitial fluid		21.0	17.7			2.8	2.25	trace
Intracellular fluid		5.4	3.0			0.5	0.75	trace

from the intestine and excreted via the kidney. This accounts for the occasional appearance of pyelograms and cystograms in human infants following the enteric administration of urokon. Separate determinations of the urokon content of intestinal and gallbladder walls revealed only trace amounts and apparently there is no storage of the compound in these organs.

The spleen, pancreas, and lungs never contained more than traces of urokon. There was no significant radioactivity detectable in the thyroids of the dogs sacrificed at one and twenty-four hours after injecting radioactive urokon. This is one indication that the iodine is tightly bound to the urokon molecule. Unreported dialysis experiments by Mallinckrodt Chemical Works likewise indicate that urokon iodine cannot be easily separated from the ring to which it is attached. No urokon radioactivity could be detected in the spinal fluid obtained from one of the dogs sacrificed at one hour.

Since the quantity of urokon recovered from the viscera did not account for all of the injected urokon, attempts were made to determine its distribution in the body fluids. The amount of urokon present in aliquots of plasma, whole blood, and muscle was determined. Using standard values for body fluid distribution in dogs (Hastings and Eichelberger[2]) and assuming the urokon to have the same concentration in all extracellular body fluids as in plasma, the distribution in the various fluid compartments was then estimated (Table IV). This is admittedly a rough estimate, but excessive refinements of this technique do not seem to be indicated. The total recovery of urokon, using this calculation, was approximately 90 per cent in all animals at one, three, five, and twenty-four hours.

DISCUSSION

In the dog the similarity of inulin and urokon clearances at plasma urokon concentrations above 30 mg. per cent leads to

the conclusion that urokon is excreted chiefly by glomerular filtration. At low plasma levels some tubular excretion can be detected. The depression of the tubular secretion of para-aminohippuric acid by the presence of urokon in the plasma is further indication that urokon does enter the cell of the tubule to a degree sufficient to obstruct tubular PAH transport and secretion. This may be by competition for the enzymatic PAH transport mechanism or by a direct toxic effect of unknown nature which depresses tubular activity in general.

Since the plasma urokon concentrations achieved in man during clinical excretory urography are relatively high (100 to 300 mg. per cent) it is obvious that the urokon contributed by glomerular filtration will far exceed that contributed by the tubules. Alterations in the quality of the resulting pyelogram will be effected only by changes in the amount of iodine filtered by the glomerulus.

The major factors altering glomerular filtration are changes in renal blood flow, blood pressure, plasma osmotic pressure, tubular hydrostatic pressure and the glomerular filtering membrane. Thus, as should be expected, clinical experience has shown that only those patients with marked impairment of effective renal blood flow (shock, vascular occlusion, or advanced renal disease) or those patients with increased tubular hydrostatic pressure such as that resulting from ureteral obstruction, show a detectable difference in the quality or timing of the nephrogram or pyelogram obtained. Josephson[3] indicates that a similar conclusion is warranted when diodrast is used as the urographic contrast medium. Although the amount of this substance contributed by tubular excretion at low plasma levels is relatively greater than in the case of urokon, it remains small compared to the amount of diodrast contributed by glomerular excretion in the presence of the high plasma diodrast concentrations usually achieved during clinical excretory urography. Thus, in either case, diodrast or urokon, the bulk of the contrast material responsible for roentgenographic visualization of the renal pelvis appears as a result of glomerular filtration. The quality of the anatomical demonstration of the renal collecting system is dependent upon the quantity of iodine in the renal pelvis at any instant, and since this results chiefly by means of glomerular filtration it is, in turn, proportional to the plasma iodine concentration.

The use of urokon results in more satisfactory visualization of the renal excretory system than was formerly possible because it has the highest iodine content per molecule and the lowest relative toxicity. Thus, higher plasma iodine concentrations can be attained with a greater margin of safety than with other commonly used media.

CONCLUSIONS

1. The presence of urokon in the plasma causes a reduction in para-aminohippuric acid (PAH) clearance roughly proportional to the injected quantity of urokon.

2. The inulin clearance (glomerular filtration) is unaffected by the intravenous injection of urokon.

3. The clearance of urokon is similar to that of inulin at plasma urokon concentrations which follow its intravenous administration in amounts equivalent to those in clinical usage.

4. It has been demonstrated that in the dog approximately 0.15 mg./min./kg. is excreted by the tubules.

5. Approximately 66 per cent of the urokon given as a single intravenous injection is excreted in the urine in five hours.

6. Approximately 20 per cent of the administered urokon is excreted through the liver, into the gallbladder and intestines. Some of this portion is subsequently absorbed from the intestines and excreted in the urine.

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ROENTGENOLOGIC DIAGNOSIS OF ANEURYSM OF THE THORACIC AORTA

WITH PARTICULAR REFERENCE TO STUDY IN THE RIGHT POSTERIOR OBLIQUE POSITION*

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DEFINITE anatomical identification of the abnormal mediastinal shadow is the most difficult problem in the diagnosis of aneurysm of the thoracic aorta; yet it is the only positive means of differentiating an aneurysm from other mediastinal masses which it may resemble. In the past, aortic aneurysm was an incurable lesion. Spectacular advances in cardiovascular surgery[1,2] have now made it possible to regard the treatment of aortic aneurysm in the same light as other thoracic tumors. Operations for the excision of aneurysms are increasing and the diagnosis is no longer only of academic interest. A simple, dependable diagnostic procedure becomes of immediate practical importance. Without recourse to the more elaborate procedure of angiocardiography, we have found study in the right posterior oblique position to be the most useful single roentgenologic technique which can furnish the information necessary for diagnosis. It is therefore surprising that this position for study of the aorta is not even mentioned in most roentgenologic textbooks.

Careful roentgenoscopic examination is indispensable. The patient stands with his back to the examiner and his left shoulder is rotated away from the screen. In so doing, the shadow of his spine moves to the left and the shadow of his heart to the right. Rotation is continued until the apex of his heart just disappears behind the vertebral column. In this position, the entire thoracic aorta is visualized. When the aorta lacks sufficient density to be clearly seen on the screen, as in younger individuals, we have found it helpful to turn the patient to and fro on a vertical axis which passes through his spine. By this maneuver, the aorta will appear to "coil and uncoil" and its shadow is readily identified. The proper degree of rotation is obtained when the apex of the heart just blends with the left border of the vertebral column (Fig. 1). In this position, the entire descending portion of the normal aorta overlies the shadow of the spine. This may, at first, appear to be an objection to the procedure. Actually, however, it contributes to diagnosis, since a descending aorta which projects beyond the spine in either direction is abnormal. Moreover, this segment of the aorta can readily be visualized by turning the patient slightly during screen examination.

Normally, as seen in the right posterior oblique position, the aortic arch lies in a horizontal plane. When this segment is oblique, it usually indicates some abnormality of the aorta itself.

The ascending aorta forms a gentle curve as it emerges from above the base of the heart. Its right border normally blends smoothly with the right cardiac border. While this smooth curve may be preserved in certain aortic lesions, the presence of a notch or incisura is abnormal, indicating angulation of the aorta most commonly due to arteriosclerosis. The ascending aorta is the widest portion of the aorta, normally measuring 4.5–5.0 cm. and it gradually tapers to the caliber of the horizontal portion where they join.

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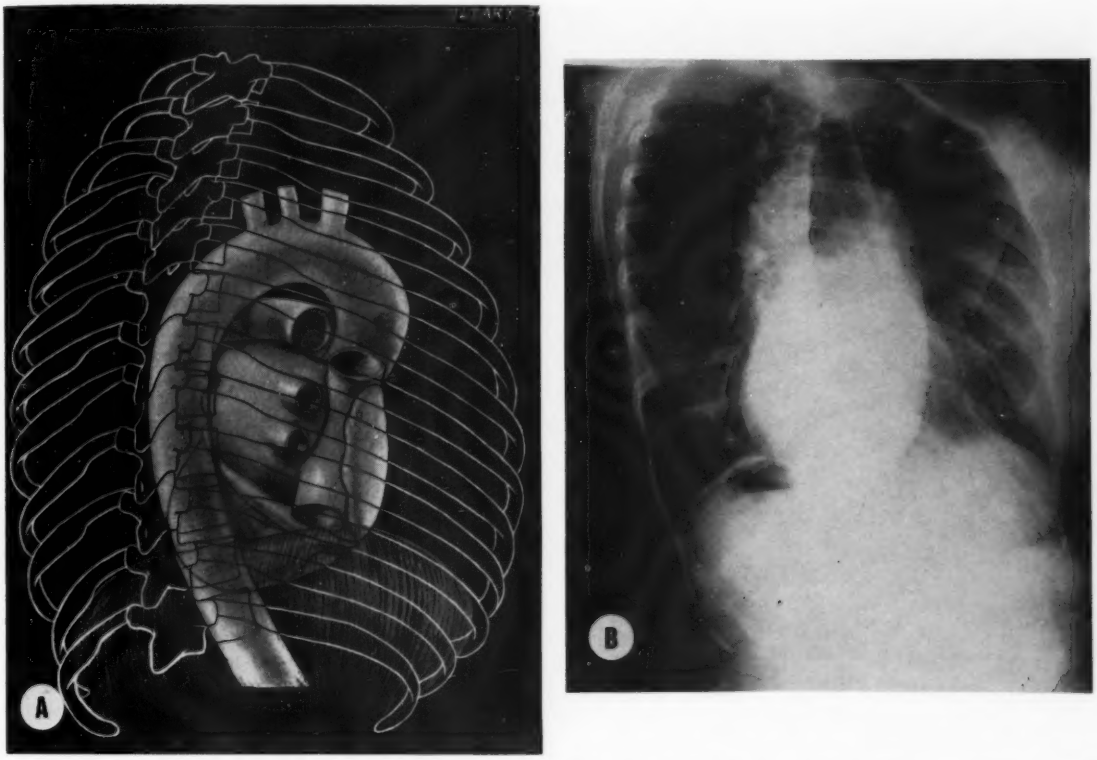

Fig. 1. (*A*) and (*B*) The normal aorta as seen in the right posterior oblique position. The correct degree of rotation is obtained when the apex of the heart just blends with the left border of the spine. The ascending aorta measures 4.5–5.0 cm., the arch is horizontal and the descending aorta overlies the spine. The latter is rendered visible by slight rotation during roentgenoscopic examination.

As seen in the frontal position, the width of the vascular pedicle is usually less than half the transverse diameter of the heart. The ascending portion does not project beyond the right border of the heart. The summit of the arch is 3.5 cm. below the sternal notch. The descending aorta is not visualized below the level of the pulmonary artery.

Widening of the aortic shadow may be due to aneurysm or other lesions which affect the aorta, such as aortitis, arteriosclerosis, hypertension, aortic insufficiency and ventricular failure. Each has certain distinctive features which help to identify it.

ARTERIOSCLEROSIS

Arteriosclerosis produces an increase in length of the aorta. This is readily seen in the frontal position, where the distance between the arch and the sternal notch is decreased (Fig. 2). Since the space within the thorax is limited, a further increase in length causes the aorta to bend upon itself. This accentuates the curve of the ascending aorta so that it appears to be wider on a film in the frontal position. The descending aorta also begins to buckle and its left border may be seen as a curvilinear shadow through the heart. It is frequently impossible, on the basis of a film in the frontal position alone, to determine whether the aortic lumen is widened or not. In a film not properly exposed, the various components of the mediastinum may blend into one continuous shadow, giving an impression of widening or aneurysm of the aorta.

In the right posterior oblique position, the arch is seen to lie in an oblique plane, the degree of obliquity corresponding to the degree of elongation of that segment (Fig. 3 and 14). The right border of the ascending

FIG. 2. Arteriosclerosis. Due to an increase in length, the arch has reached the sternal notch.

aorta joins the cardiac outline with a sharp incisura or notch due to buckling of the aorta. It is this buckling which produces widening of the aortic shadow in the frontal position. The greatest changes, however, are frequently seen in the descending aorta. Various degrees and types of buckling due to elongation of the descending portion may cause it to project markedly to the left or right of the spine or assume an "S" curve (Fig. 4 and 14). In any case, it can be seen that while there is an increase in the length of the aorta, its caliber is not changed. This is an important observation, since study in the frontal position may give an impression of aneurysmal dilatation. As observed in the right posterior oblique position, arteriosclerotic calcification is more commonly found in the inferior wall of the arch; luetic calcification is more often found in the superior wall of the ascending portion (Fig. 5 and 14). In the frontal position, arteriosclerotic calcification is most commonly seen in the "knob." In advanced cases it may be seen in the ascending and descending aorta.

Roentgenoscopic examination furnishes additional information by study of aortic pulsations. While aortic pulsations may, at first, appear to show normal or increased

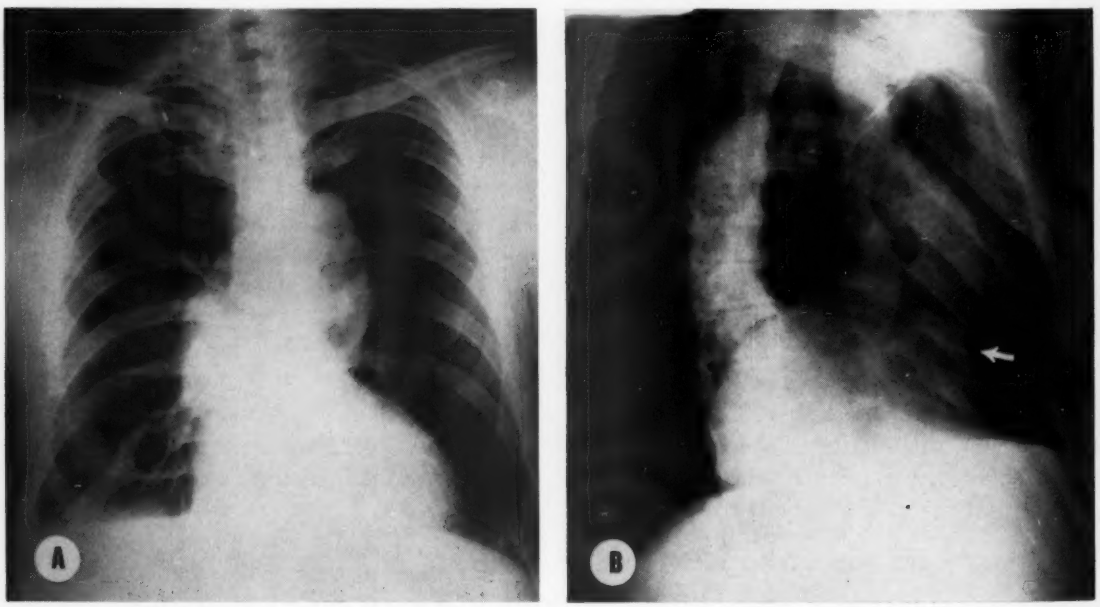

FIG. 3. Arteriosclerosis. (*A*) The widened supracardiac shadow cannot be defined and its cause is uncertain. (*B*) Right posterior oblique position. The prominence of the ascending aorta is due to buckling. Note the sharp incisura at the cardiovascular junction (arrow). Note obliquity of the arch. This is usually proportionate to the degree of sclerosis.

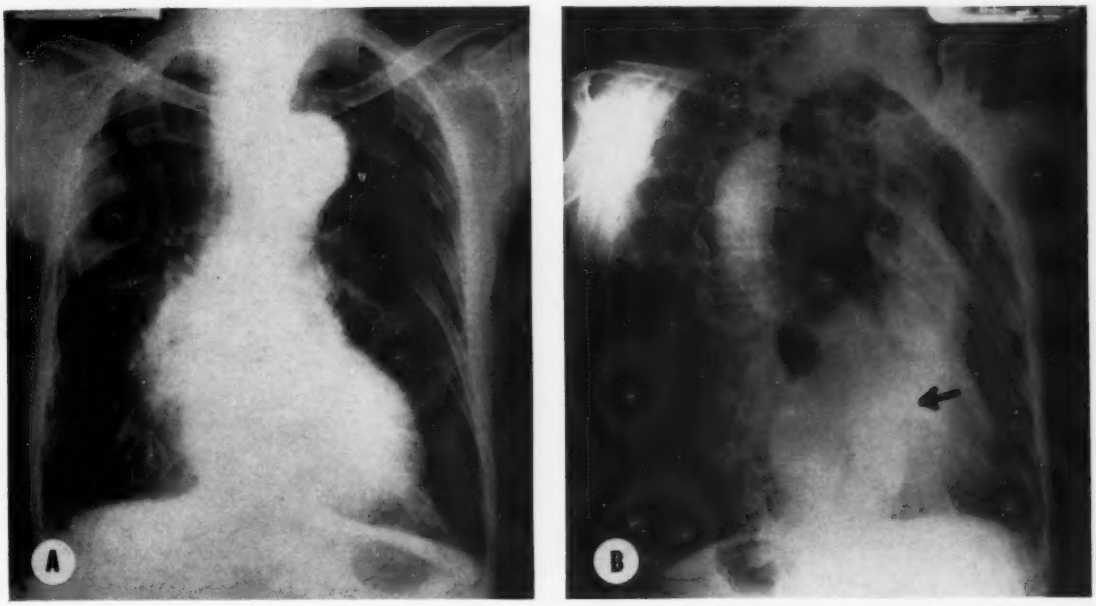

Fig. 4. Arteriosclerosis. (*A*) Note the marked increase in length of the aorta. The area of density at the right cardiac border cannot be definitely identified. (*B*) Right posterior oblique position. The descending aorta forms an "S" curve. Bowing of the lower descending portion (arrow) is responsible for the prominent right cardiac border in (*A*). Note marked obliquity of the arch.

amplitude, careful examination will usually reveal a mass movement of the aorta, in which the whole segment shifts, rather than expands. This type of movement is quite

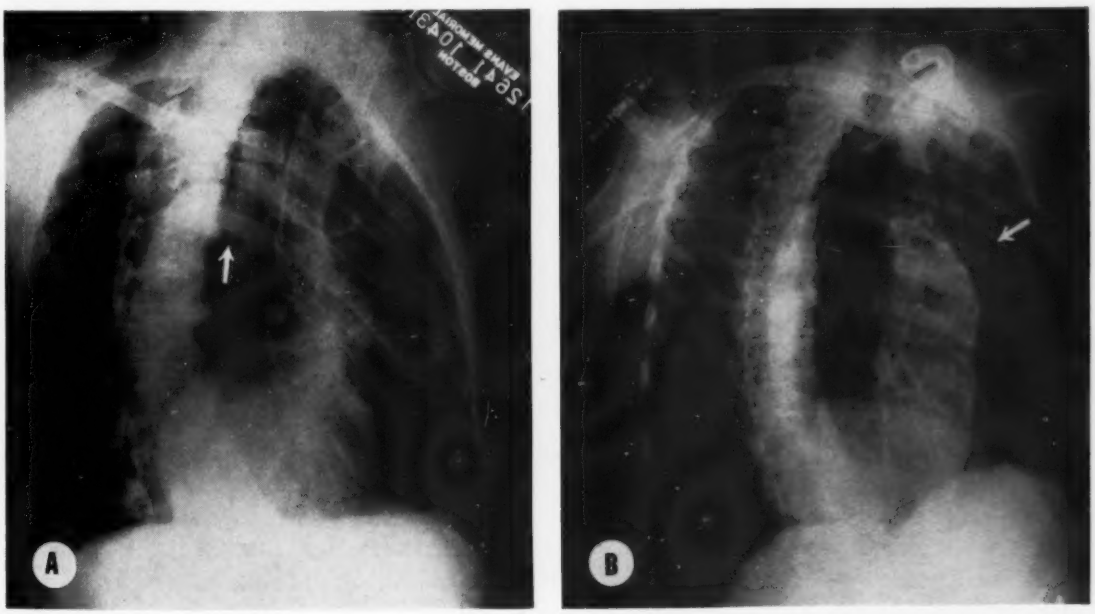

Fig. 5. Roentgenograms in right posterior oblique position showing: (*A*) Arteriosclerosis. Note obliquity of the arch, the notch at the cardiovascular junction and a calcified plaque in the inferior wall of the distal portion of the arch (arrow). (*B*) Luetic aortitis. The horizontal direction of the arch is fairly well preserved. Note how smoothly the ascending aorta blends with the cardiac contour. There is calcification in the superior wall of the ascending portion (arrow).

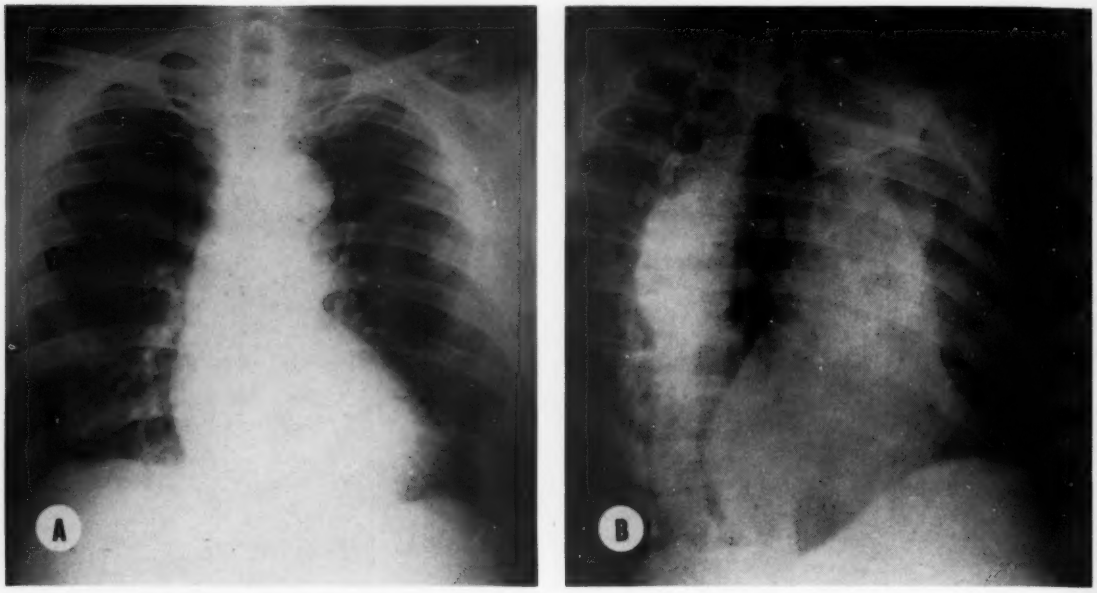

FIG. 6. Luetic aortitis. (*A*) The aortic shadow is widened. The smooth curve of the ascending aorta is diminished or irregular. The "knob" usually becomes less prominent. (*B*) Right posterior oblique position. The widened ascending aorta blends smoothly with the contour of the heart. The arch and beginning descending portion are also widened.

different from that observed in hypertension, aortic insufficiency or aortitis.

AORTITIS

One of the earliest changes in aortitis is inflammation of the small lymph vessels and lymph spaces that accompany the vasa vasorum. The inflammatory process excites a periarteritis and ultimate thrombosis of the small vessels. As a result, there is a loss of tone and destruction of muscle fibers and fragmentation of elastic fibers. These elements are soon replaced by noncontractile scar tissue. Scar tissue deposited in the media as a result of mesaortitis also extends into the adventitia to cause irregular thickening of that coat. Hwiliwitzkaja[3] has shown that, in maintaining the tone of the aorta, the muscle fibers play a more important role than the elastic fibers and that the latter exert their influence only after the muscle fibers are relaxed. Thus, there is early impairment of the tone and substance of the muscle fibers. This, together with further loss of elastic recoil resulting from the destruction of elastic fibers, allows the aortic walls to relax and its lumen to become widened. Actual thickening of its walls also contributes to an increase in the width of the aorta.

In the frontal position, the curve of the ascending aorta becomes diminished and the "knob" less prominent. At times, minute irregularities in the contour of the ascending aorta, due to fibrous tissue scarring, may be seen (Fig. 6). Pulsations of the aorta are diminished. In the right posterior oblique position widening of the ascending portion is best demonstrated, for, in this position, one can differentiate an actual increase in caliber from apparent widening due to buckling at the cardiovascular junction. Calcification in the ascending aorta alone, usually seen in the superior wall in the right posterior oblique position, is almost invariably of luetic origin.

HYPERTENSION

The aorta usually shows abnormal changes in hypertensive disease.[4] There

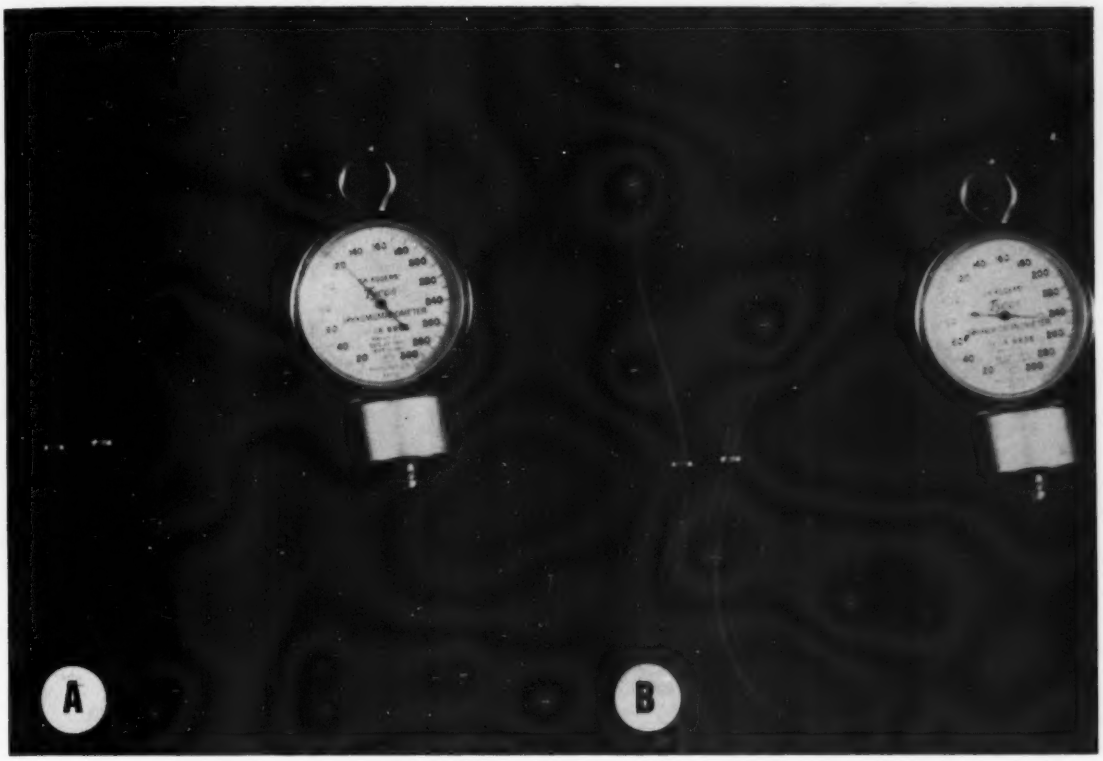

Fig. 7. Simple experiment to demonstrate the effect of increased pressure in an elastic coil having two fixed points. (*A*) Pressure of 120 mm. mercury. (*B*) Pressure of 240 mm. mercury. Note how the loop "uncoils" and widens.

may be actual widening of the aortic shadow or an increase in length and tortuosity of the aorta commonly associated with arteriosclerosis, or both. We have observed these changes in young, as well as older adults.

In the frontal position the widened aortic shadow may be mistaken for aneurysmal dilatation or aortitis. On roentgenoscopic examination, the cause of the widened shadow is frequently found to be increased pulsatile movement of the aorta. This is more marked in patients having a large pulse pressure and is probably due to the increased hydrodynamic thrust in systole associated with some yet unexplained stimulus from the autonomic system (Fig. 7). We have termed this "functional widening" of the aorta, since the aortic shadow becomes narrower after sympathectomy.

Study in the right posterior oblique position permits better visualization of the aortic caliber and movement. The increased tortuosity of the aorta is clearly demonstrated and the type of pulsations differentiates this functional widening from aortitis and arteriosclerosis.

AORTIC INSUFFICIENCY

The aortic shadow is frequently widened in aortic insufficiency. The latter may occur in the course of luetic, rheumatic or hypertensive heart disease. The vascular pedicle is frequently widened in ventricular failure. Brief consideration of these changes may be useful in differential diagnosis. In any case, aortic insufficiency is accompanied by dilatation of the left ventricle. This is usually apparent on screen examination and is readily verified by study of the roentgenogram. Typically, the aorta shows a collapsing type of diastole ("water-hammer") in

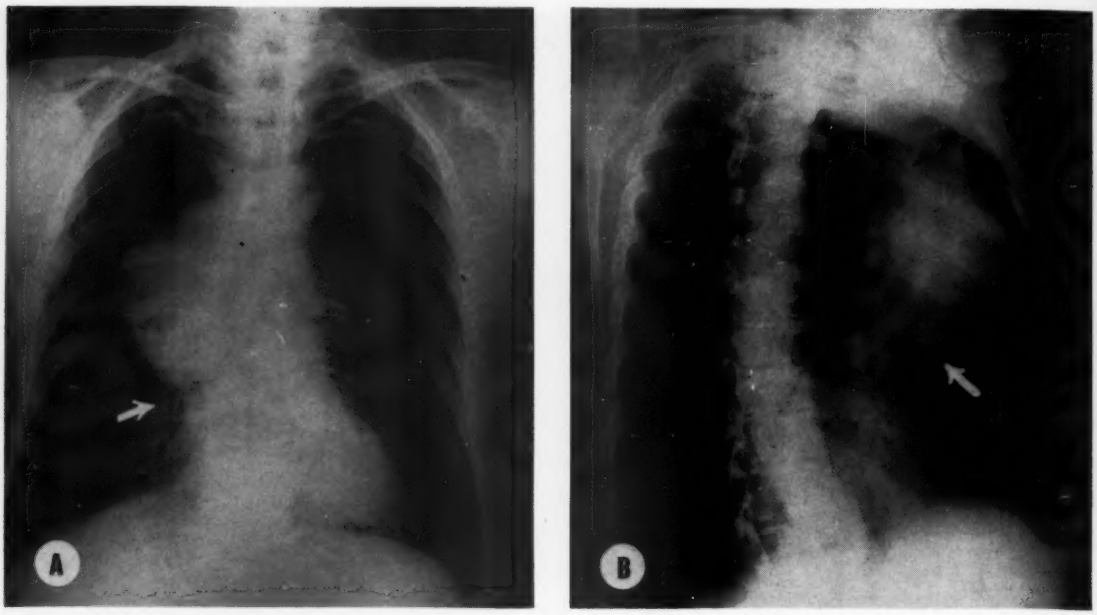

FIG. 8. (*A*) and (*B*) Mass in right mediastinum resembling aneurysm of ascending aorta. Note notch (arrow) at junction of mass and heart shadow in both views. In the right posterior oblique position (*B*) the mass can be separated from the aorta. The arch is oblique due to arteriosclerosis, but there is no widening of the aortic lumen. These findings exclude aneurysm. The tumor was a thymoma.

which narrowing of the aortic caliber is quick and sharp. The appearance is typical and almost pathognomonic of aortic insufficiency and is more pronounced as the pulse pressure increases. We have, however, observed this type of contraction in thyrotoxicosis[5] (in which the pulse pressure is frequently high).

The wide vascular pedicle seen in ventricular failure is due, largely, to widening of the superior vena cava. Frequently, however, there is associated widening of the aorta. In these cases, the superior vena cava is recognized as a more or less straight line which emerges from the shadow of the ascending aorta just above the cardiovascular junction and which, on roentgenoscopic examination, shows faint lateral pulsations which are synchronous with ventricular systole. Widening of the aorta, in these cases, is frequently due to a relative insufficiency resulting from stretching or relaxation of the aortic ring. Further evidence of ventricular failure may be found in demonstrable enlargement of the right ventricle, pulmonary congestion, fluid in the pleural cavity or interlobar fissures and alteration of the cardiohepatic angle.[6]

ANEURYSMS

Aneurysms of the thoracic aorta are most commonly of luetic origin. They may be fusiform, involving evenly all walls of a long aortic segment, or saccular, in which localized outpouching of one wall predominates. In the order of frequency, aneurysms are found most often in the ascending aorta. This is probably due to the fact that the earliest lesions of aortitis occur in the first 5 centimeters of the ascending aorta.[8] Next in frequency is involvement of the arch. Aneurysm of the descending portion is least common.

The diagnosis of aneurysm depends upon the demonstration of an abnormally widened aortic segment, and it is common knowledge among roentgenologists that this can be an exceedingly difficult problem. There are, however, certain roentgenologic signs that are helpful. The presence of an aneurysm might first be suspected from examination of a routine film of the chest.

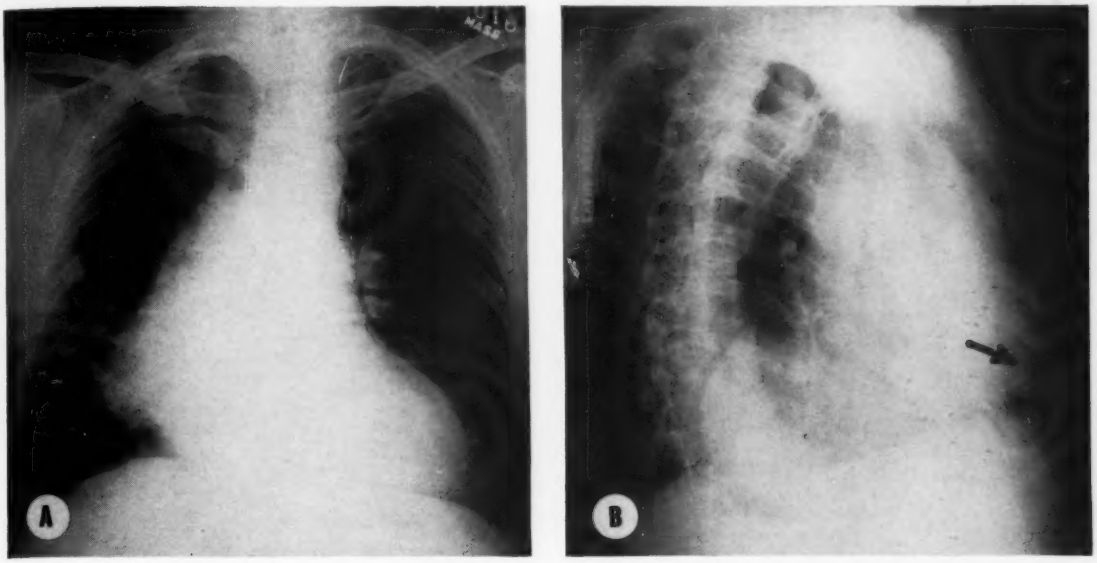

Fig. 9. Luetic aneurysm involving sinus of Valsalva; aortic insufficiency. (*A*) The wide mediastinal mass blends smoothly with the heart shadow. Bulging of the right auricular segment is due to sinus involvement. The left ventricle is also enlarged. (*B*) The bulging right auricle (arrow) and marked dilatation of the ascending aorta are well demonstrated.

In the posteroanterior view the shadow of the abnormal mass is always continuous with the mediastinal shadow. The trachea may be displaced or compressed, but atelectasis is an uncommon finding. There may or may not be erosion of ribs, sternum or vertebrae. The heart may be depressed by an aneurysm of the arch, but it is not enlarged unless there is aortic insufficiency or other associated heart disease. When the lateral border of the abnormal shadow blends in a smooth, continuous curve with the shadow of the mediastinal structures, it favors the presence of aneurysm. Conversely, when the junction of these shadows is marked by a notch or incisura, the possibility of tumor is more likely (Fig. 8 and 14). On roentgenoscopic examination, the presence or absence of "expansile pulsations" is often disappointing. Since many aneurysms contain a large, organized clot, pulsation is frequently diminished. Moreover, tumors firmly attached to the aorta may show transmitted pulsation, and considerable experience may be necessary to differentiate autochthonous from transmitted pulsation. It may, perhaps, be more useful to observe whether or not the mass moves on swallowing. While some tumors attached to the esophagus or trachea move on deglutition, an aneurysm never does.

The right posterior oblique position affords the best method of localizing the enlarged aortic shadow. In this position, an aneurysm of the ascending portion will appear just above the cardiovascular junction and the widened shadow will approach the right thoracic wall. An aneurysm which involves the beginning of the ascending aorta is frequently associated with aortic insufficiency. Sufficient evidence for diagnosis is obtained when one finds an aneurysm in this region together with enlargement of the left ventricle. Aneurysms which involve the sinuses of Valsalva frequently project into the right auricle. Films in the posteroanterior and right posterior oblique position will show abnormal bulging of the right auricular segment of the heart shadow (Fig. 9 and 14). An aneurysm of the arch will show as a mass, continuous with the aortic shadow, extending cephalward (Fig. 10 and 14). Aneurysm of the innominate artery is also well demonstrated in this view. An aneu-

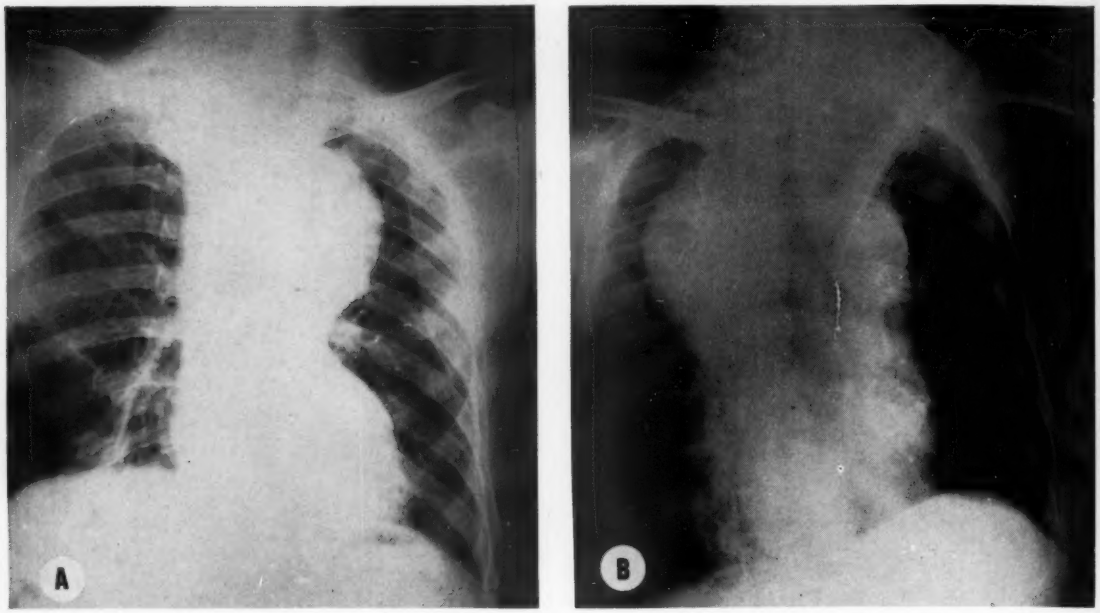

FIG. 10. Luetic aneurysm. (*A*) There is a saccular aneurysm of the descending aorta and of the innominate artery. Note marked displacement of the trachea. The density in the right upper mediastinal and cervical regions is due to aneurysm. There is no evidence of atelectasis. (*B*) Right posterior oblique position localizing the dilatations. Note calcification in the superior wall of ascending aorta. The right first rib has been eroded. There was fatal hemorrhage from external rupture of innominate aneurysm.

rysm of the descending aorta will project beyond the shadow of the spine (Fig. 11 and 14). Calcification in the aneurysm is readily localized and we have frequently seen it in the right posterior oblique position when it was not discernible in the frontal posi-

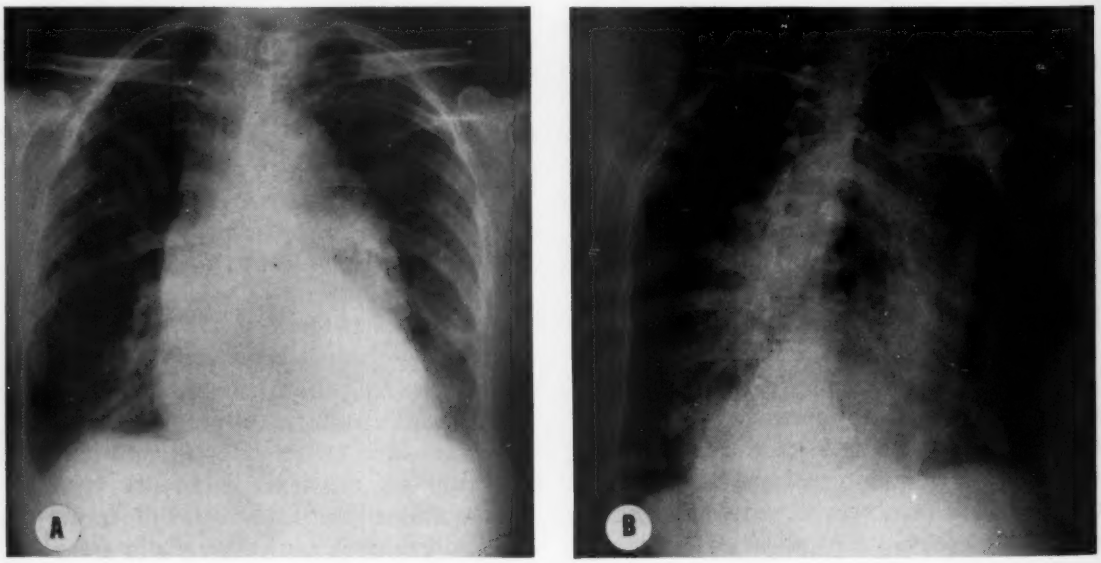

FIG. 11. Aortic aneurysm. (*A*) The widened mediastinal shadow was interpreted elsewhere as "bronchogenic carcinoma." (*B*) Right posterior oblique position showing marked projection of descending aorta beyond the spine due to dilatation. The arch and ascending portions are also dilated.

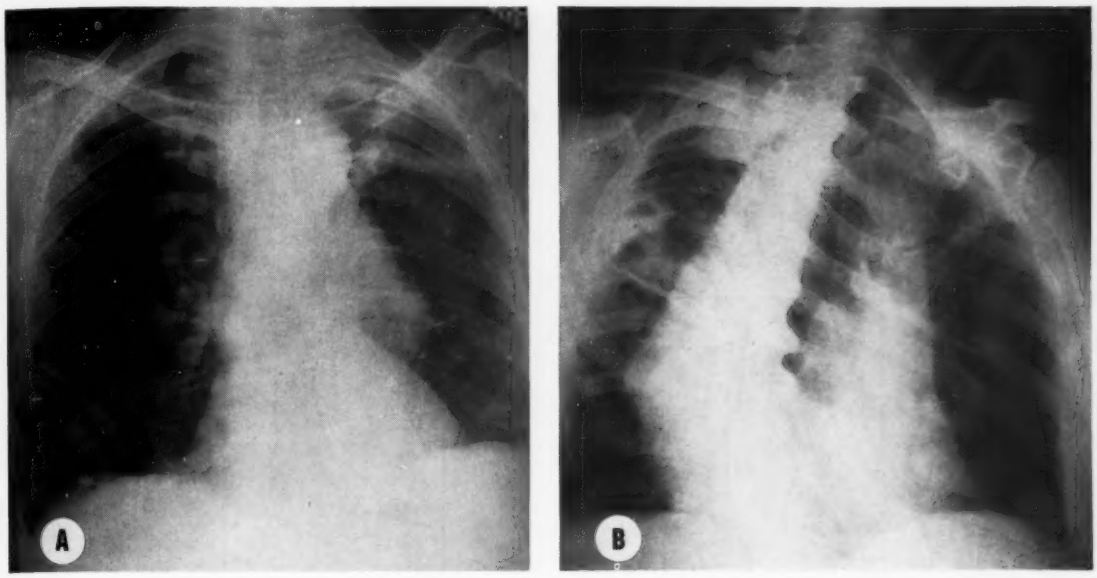

Fig. 12. Dissecting aneurysm with rupture into the pleural cavity. (*A*) Note the straight borders and sharp angulation of the descending aorta. (*B*) The straightened and angulated descending aorta shows numerous knobby irregularities due to collection of blood clots in the media.

tion. Regardless of the location of the aneurysm, the entire thoracic aorta should be carefully studied, for aneurysm is a disease of the aorta and changes of varying degree are found throughout its entire length. For example, widening of the beginning of the ascending portion helps to confirm the diagnosis of early fusiform aneurysm of the descending portion. This was formulated as a "rule" by Thoma and Kienböck who stated

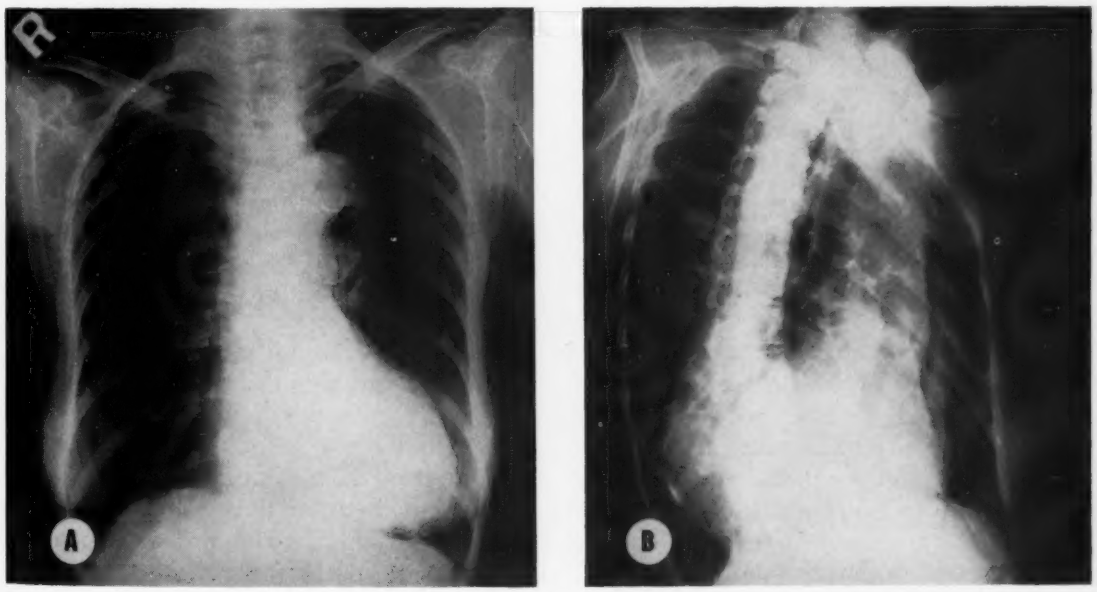

Fig. 13. Dissecting aneurysm of descending aorta which re-established continuity with the aortic lumen. (*A*) Posteroanterior view six years after an attack of excruciating pain which radiated from the sternum straight through to the back, associated with collapse. (*B*) Right posterior oblique position showing the straightened and angulated border of the descending aorta. There is calcification of the intramural clot.

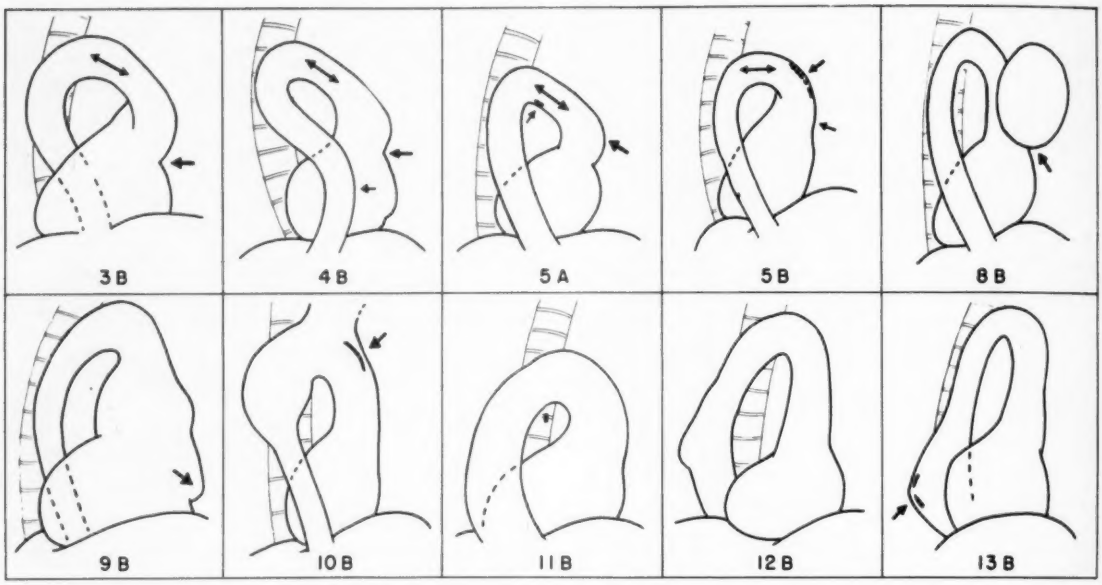

FIG. 14. 3B: Arteriosclerosis. Note obliquity of arch, and notch. Buckling of ascending portion produces prominent right supracardiac border in Figure 3. 4B: Arteriosclerosis. Note obliquity of arch, and notch. Bowing of descending portion produces prominent right cardiac border in Figure 4. 5A: Arteriosclerosis. Note obliquity of arch, notch and calcification in inferior wall. 5B: Luetic aortitis. Note horizontal direction of arch, smooth cardiovascular junction and calcification in superior wall. 8B: Thymoma. Note incisura where the upper and lower poles join the cardiovascular shadow. The normal caliber of the aorta is preserved. This excludes aneurysm. 9B: Aneurysm of ascending aorta and sinus of Valsalva, aortic insufficiency. Note bulging right auricle. 10B: Luetic aneurysm localized by right posterior oblique position. Note calcification in superior wall, involvement of descending portion, arch and innominate artery. 11B: Right posterior oblique view demonstrates aneurysmal dilatation of entire thoracic aorta. 12B: Dissecting aneurysm. The true nature of the lesion is clearly defined. Note angulation and knobby irregularitie2 o! descending aorta. 13B: "Healed" dissecting aneurysm. Appearance almost identical with 12B except for calcification of intramural clot. Film six years after dissection occurred.

that localized aneurysmal enlargement hardly ever occurs in an aorta that is otherwise free of disease.

DISSECTING ANEURYSM

Dissecting aneurysm is far more frequently associated with arteriosclerosis than syphilis. The lesion first appears in the media. Atrophy of elastic lamellae, probably associated with necrosis of muscle fibers, allows the coat to relax, so that hemorrhage from the vasa vasorum splits the coat longitudinally. Dissection also extends circumferentially, so that two distinct tubes, one within the other, may be formed. There may or may not be a tear in the intima. Dissection may extend from the semilunar valves to below the bifurcation of the abdominal aorta. Dissection may extend through the external coat, with fatal hemorrhage into the pericardium, heart, pleura or mediastinum, or it may tear through the intima to re-establish continuity with the aortic lumen, or it may be arrested by the accompanying fall in blood pressure.

In general, the most striking feature in the appearance of a dissecting aneurysm is the straightening of the aortic curve in the involved segment. The straightened aortic wall usually shows numerous localized knobby irregularities due to a larger collection of blood clot at these sites. The aortic caliber is widened. The widened segment swings back to the mediastinum, not in a curve, as is the case with ordinary aneurysm, but at an angle. The appearance of dissecting aneurysm on a posteroanterior roentgenogram has not infrequently been

mistaken for mediastinal tumor. Examination in the right posterior oblique position should establish the diagnosis in most cases (Fig. 12 and 14). If the ascending aorta is involved, pulsations in this region are markedly diminished. Not infrequently, dissection may occlude the superior mesenteric or common iliac arteries. If continuity with the aortic lumen is re-established and further dissection has ceased, the lesion is, for the time, arrested (Fig. 13 and 14). Osler[7] has described this as "the most remarkable reparative process seen in the human body."

SUMMARY

The roentgenologic diagnosis of aortic aneurysm is frequently a difficult problem. Not only must it be differentiated from mediastinal tumors, but from other acquired lesions of the aorta as well. Roentgenologic study in the right posterior oblique position is presented as the most useful single method of establishing a diagnosis without recourse to the more elaborate technique of angiocardiography.

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DISCUSSION

Dr. John W. Kirklin, Rochester, Minnesota. I am certainly not qualified to comment on the strictly roentgenologic aspects of this excellent presentation by Dr. Levene. Nonetheless, I am happy to be able to say a few words in regard to it since, as a surgeon, I welcome all contributions which result in simple and yet precise methods of diagnosis.

Dr. Levene has emphasized the need for more accurate methods of diagnosis since surgical treatment of aortic aneurysms, whatever their location, is feasible at the present time. De Bakey and Cooley, and Bahnson have been largely responsible for the present aggressive policy of resection which we and others now practice, although numerous advances in cardiovascular and general thoracic surgery have contributed to the growth of this method of treatment. There can be little question but that an aggressive policy is advisable, for the poor prognosis of all aortic aneurysms is notorious.

In the past, many forms of surgical therapy have been utilized for aortic aneurysms but none of these has met with success in a high percentage of cases. For this reason and because of the practicability and theoretical merit of resection of aortic aneurysms, we have abandoned all forms of treatment other than resection unless the patient's general condition precludes it. It is true that sufficient data have not accumulated to allow us to state with assurance the long-term state of cases treated by resection but the results to date are highly encouraging.

Aortic aneurysms present a somewhat more formidable problem than most aneurysms in the extremities, for the aortic pathway must be preserved or reconstructed in conjunction with the aneurysm resection. In cases of arteriosclerotic aneurysm of the abdominal aorta, this is best done by the insertion of a preserved homologous aorta graft after resection of that portion of the abdominal aorta containing the aneurysm.

Aneurysms arising from the aortic arch or the upper descending aorta must be approached in a somewhat different fashion. Cross-clamp-

ing of the aorta at these levels is not well tolerated. Future developments in cardiovascular surgery may alter this situation. At present, at least, resective surgery can be used in these areas only for saccular aneurysms, which fortunately are the most common. After control of the neck of the aneurysm is obtained, the aneurysm can be resected and the resultant defect in the aortic wall closed by suture—the so-called "lateral aortorrhaphy."

The surgical approach to aneurysms of the aortic arch and descending thoracic aorta varies according to the location of the aneurysm (slides).

From these remarks, it can be seen that more precise information is required now than was necessary before resection of these aneurysms was feasible. The exact origin of the aneurysm must be ascertained and a distinction must be made when possible between saccular and fusiform aortic aneurysm. The exact information gained by Dr. Levene from his roentgenologic studies in these cases is thus of extreme importance to all interested in this subject.

I wish to express my appreciation to members of this Society for the privilege of discussing Dr. Levene's fine contribution.

Vol. 72, No. 6

ROENTGEN DEMONSTRATION OF CERVICAL NERVE ROOT AVULSION*

By JOHN E. WHITELEATHER, M.D.
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CERVICAL nerve roots may be avulsed from the cord or torn apart in the intervertebral foramina or immediately outside by injuries that impose severe traction upon the brachial plexus. Such injuries may occur when strong downward pressure is applied to the shoulder with the head fixed or pushed in the opposite direction. The degree and extent of the motor and sensory deficit, of course, depends upon the number of roots involved and whether all fibers of a root are avulsed or separated. If roots are avulsed, or torn within the foraminal canal, the injury is irreparable. However, if it should be located just outside the foramen, there is some hope that neurorraphy might be successfully done.

Clinical diagnosis of these injuries is not too difficult and is based upon the history and neurological findings. Exact localization of the site of injury to an individual nerve is more difficult and apt to be complicated by soft tissue and osseous trauma in recently injured patients. When Horner's syndrome is present the first thoracic root is involved and if the muscles mediated by the long thoracic and dorsal scapular nerves are powerless, a higher lesion is indicated. There can be a combination of avulsion, extraspinal separation and plexus injuries which may present a most confusing neurological puzzle.

Myelography offers a quick, relatively easy and accurate method of demonstrating avulsion or root tear within the foramen. By exclusion, the remaining lesions could be considered extraspinal. Myelography has the additional advantage that it can often be employed early, even when fractures or soft tissue lacerations hinder or prevent accurate neurological evaluation.

The roentgen diagnosis of root avulsion was first reported by Murphey, Hartung and Kirklin,[1] who described the myelographic findings in 1 patient seen in an army hospital. Later, they studied 2 additional cases, which they explored, and correlated the roentgen findings and surgical pathology. These cases, plus some of the present group, have since been reported by Murphey but are not yet published. All of the present group of patients are from the private service of Drs. Semmes, Murphey and associates and were seen at the Baptist Memorial Hospital. The interval between the injury and the myelographic study varies from three weeks to fifteen years.

Standard cervical myelographic technique is employed using 6 cc. of warmed pantopaque. The spinal needle is left in place to facilitate removal of the oil. Recently, we have used 9 cc. on some occasions, finding that it is easier to manipulate, fills the theca better and is no more difficult to remove. The abnormalities are of two types and both are usually seen in the same patient. The *first* and most arresting are one or more extradural collections of oil in pockets or pseudodiverticula. These lie along side or project into the intervertebral foramina and represent a tear or rent in the dura left by the avulsed root. A few fibers may still remain in these pockets. The *second* is less conspicuous but indicates a complete avulsion. The dura has healed, sealing the rent and leaving a smooth straight contour or only a tiny notch where the normal root shadow should be.

REPORT OF CASES

CASE 1. R.B., a fourteen year old boy, walked into a spinning airplane propeller and was struck on the left shoulder. He sustained fractures of the clavicle, and humerus, soft

* Presented at the Fifty-fourth Annual Meeting, American Roentgen Ray Society, Cincinnati, Ohio, Sept. 29-Oct. 2, 1953.

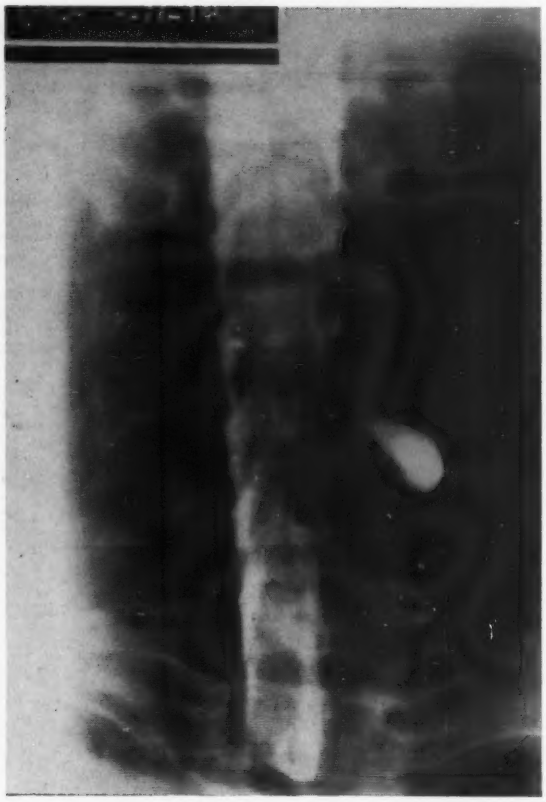

Fig. 1. Case I. Extradural pocket at 6th interspace, large defect at 7th, small defect at 1st thoracic.

tissue lacerations and immediate paralysis of the left arm. There was also a Horner's syndrome. He was referred for neurological consultation six months after the accident.

Myelography showed normal 5th and 6th nerve roots. At the 6th interspace, an extradural pocket filled with pantopaque and extended through the foramine, indicating probable avulsion of the 7th root. An irregular filling defect obscured the 8th cervical and 1st thoracic roots and root sleeves (Fig. 1). It was felt that this was due to complete avulsion of the roots with cicatricial sealing of the dural rents. The left diaphragm was observed to be paralyzed.

Because of the child's pathetic condition, it was decided to explore the extraspinal trunks in the hope that the injury to the 5th and 6th nerve roots might be reparable. The 5th root was intact but a large neuroma involved the lateral branch. The 6th root was fibrosed and hard, on into the foramen. The 7th root appeared similar but in freeing it, the pocket, extending through the foramen was inadvertently opened, releasing a small quantity of clear cerebrospinal fluid. No nerve fibers were found. The 8th cervical and 1st thoracic were absent in the foramina, the ends lying about 2 cm. laterally. Because of poor surgical condition, the operation was terminated.

Case II. D.K., female, aged thirty-nine, was injured in an automobile accident six weeks previously and was unconscious for seven days. Upon regaining consciousness, she was unable to move the right arm or flex the elbow. She suffered considerable pain in the neck and shoulder but she had also had neck pain before the accident. Neurological examination revealed a segmental paralysis of muscles mediated by cervical nerve roots 5 and 6 and incomplete sensory loss of C_4, 5 and 6. Since there were some signs and symptoms of a ruptured disc, as well as root avulsion, myelography was advised. This procedure did not demonstrate any extradural pocket but did show a loss of normal root niche of the 4th and 5th roots (Fig. 2). A hemispherical indenture, somewhat resembling a herniated disc, was located opposite the 5th foramen but was thought to be too large for a disc defect, and probably represented

Fig. 2. Case II. Blunted root notch at 4th right interspace, herniated disc defect at 6th.

an avulsion with cicatrix. The 7th and 8th roots appeared very small and may have been partially avulsed. The right diaphragm was paralyzed.

Exploration was carried out. Only the 5th and 6th roots were explored and both were avulsed with no remaining fibers. There was a hard bar of disc material at the 5th interspace which had apparently been present for a long time. The root which normally would have passed over it was avulsed and nothing further was done. She was discharged unimproved.

This case well demonstrates that several types of pathology can be present to confuse the picture.

Case III. R.C., a twenty-two year old female, was thrown from a car, suffering head and left upper extremity injuries, including a fracture of the humerus. There was complete motor and sensory deficit of the arm and shoulder, and a Horner's syndrome. After recovery from the head injury and healing of the fracture, she was referred for neurosurgical evaluation. Myelography demonstrated obliteration of the root notch at the 6th foramen and extradural pockets at the 7th and 1st thoracic, indicating avulsion of the 7th and 8th cervical and 1st thoracic roots (Fig. 3). Exploration was not advised.

Fig. 3. Case III. Small blunted root notch at left 6th, large extradural pockets at 7th and 1st thoracic interspaces.

Fig. 4. Case IV. Large extradural sacculation at the 6th and 7th foramina.

Case IV. J.G., male, aged forty, was struck on the shoulder by a falling pipe fifteen years before. There was almost complete motor and sensory loss of the shoulder and extremity. Later, a severe causalgia of the finger and thumb developed, for which a sympathectomy was done in another city. The pain was not relieved and further motor deficit occurred. His brachial plexus had been explored at still another hospital and, though somewhat fibrosed, was pronounced intact.

Myelography was advised and carried out which showed a large extradural sacculation projecting through the 6th and 7th foramina, interpreted as a complete avulsion of the 7th and 8th cervical roots (Fig. 4). Since most of the oil entered these pockets, the condition

FIG. 5. Case v. Blunted root sleeve at 6th interspace (left) and extradural pocket at 7th interspace.

of the other roots could not be ascertained. Surgical exploration was not advised.

CASE V. O.T.C., a sixty-four year old man, received a severe injury to the left shoulder while blasting stumps. He was seen shortly afterwards. He had a large axillary hematoma, extensive soft tissue lacerations of the forearm, motor and sensory deficit of the hand and fingers and a Horner's syndrome. The arm was not involved neurologically.

Myelography was done three weeks later and showed a sealed over 7th root sleeve and an extradural pocket projecting through the 7th intervertebral foramen (8th root) (Fig. 5). On other views, a smooth dural bulge obliterated normal markings at the 1st thoracic root site. Surgery was not advised and there has been no subsequent improvement.

CASE VI. D.F.E., a seventeen year old girl, fell from a moving car, two and one-half years previously, landing upon her right arm and shoulder. Except for some preservation of sensation of the 5th and 6th cervical roots, the right arm was paralytic. There was an incomplete Horner's syndrome. Myelography showed a large extradural collection of opaque medium alongside the cord at the 6th foramen and a smaller one at the 7th (Fig. 6). The normal notch was sealed over at the 1st thoracic. Interpretation was 7th, 8th cervical and 1st thoracic avulsion. Surgery was not advised.

CASE VII. J.W.C., a four year old boy, injured in a car accident three months before admission. He had been unconscious for seventeen hours and had sustained a fracture of the right clavicle, head of the right humerus and right femur. He was found to have complete loss of power in all muscles of the shoulder girdle except the serratus magnus and a portion of the pectoralis major. The arm was powerless except for the extensors of the forearm and fingers and supinators of the forearm. Sensation was absent in the same regions.Myelography showed a tremendous outpouching at the right 7th intervertebral foramen and another pocket lying along the theca upward as far as the 5th foramen (Fig. 7). Thus, it was felt certain that

FIG. 6. Case VI. Large extradural pocket at right 6th and small one at 7th interspace.

the 6th, 7th and 8th roots were avulsed. No attempt at repair was advised.

Case VIII. W.P.M., male, aged fifty, was struck on the head and left shoulder, five months previously, by the block of an overhead crane. He was unconscious for thirty hours and, upon regaining consciousness, found he was unable to move his left arm. Since he had a fractured humerus, little attention was paid to the paralysis at the time. Arriving for neurosurgical evaluation, he was found to have complete paralysis of the shoulder girdle and loss of sensation in the arm and hand except for the medial two digits and the region mediated by the 2nd dorsal root. Myelography was carried out and demonstrated an extradural pocket, indicating avulsion of the 6th cervical root with small notches representing the 4th, 5th, 7th, and 8th (Fig. 8). It was felt that these were either partially or completely torn also. Surgery was not advised.

Fig. 8. Case VIII. Small extradural pocket at 5th foramen. Fourth, 5th, 7th and 8th root sleeves narrowed and contracted.

SUMMARY

We have had the opportunity of personally examining nine instances of cervical root avulsion, plus the roentgenograms and case histories of four others. Two of this series and three of Dr. Murphey's original military cases have been explored. In all instances, one or both of the myelographic abnormalities, an extradural collection of opaque medium, or a blunted root notch, have been present. In view of the surgical correlation, the findings may be considered practically pathognomonic, particularly when associated with the history of trauma and the proper neurological deficit.

In none of the cases that have been operated upon with the hope of surgical restitution has it been possible to effect any improvement. The value of the myelographic procedure is simply to confirm the diagnosis and to present evidence contraindicating the advisability of surgery which up to now has been fruitless.

Fig. 7. Case VII. Large sacculation covering the 6th, 7th and 8th nerve roots.

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DISCUSSION

DR. HAROLD O. PETERSON, St. Paul, Minnesota. My first reaction, on reading the title of Dr. Whiteleather's paper, "Roentgen Demonstration of Cervical Nerve Root Avulsion," was that it can't be done. In my simple way I imagined he was demonstrating the actual nerve trunks, and I just couldn't figure out how it could be done.

I am convinced now, however, that he has described a distinct entity which I was formerly completely unaware of. After seeing his illustrations about a month or so ago, I was able to find 4 cases in the records of the University Hospitals which fit this roentgenographic picture, but only one of these had an injury. It is not surprising, therefore, that as these 4 cases came along we had not been impressed with the association of the extradural "cyst" and the injury. We simply called those cases "arachnoid cysts."

Two of our cases were in the cervical region, and 2 in the thoracic. One of the cervical cases had a very severe injury twenty-one years earlier, and had had a semiflail arm ever since. He came in for study because of a severe pain in the shoulder and arm of six months' duration. When we demonstrated this so-called "arachnoid cyst" we felt that it was the cause of his pain. He was explored and the cyst was removed, and he was relieved of pain. He had no improvement in the motor weakness, but the pain of which he complained was relieved.

The other 3 cases which we have had, in which there was no injury, also were operated on. Arachnoid cysts were found in each case, but in one of the thoracic cases the cysts were multiple, and in that case there was bone erosion of the pedicles in several areas in the thoracic spine. From a roentgenographic standpoint, therefore, we have a similar condition which is not produced by trauma.

It would seem possible to me that one could have a cyst of this type with such a small opening that the contrast medium might not enter the cyst. In such a case the extradural pocket lying alongside the dural sac would produce a flattening or deformity of the dural sac, which would simulate the deformity produced by scar tissue as Dr. Whiteleather has pointed out.

In the future, in view of Dr. Whiteleather's paper, I believe we can alert our clinicians about this condition, and do more myelograms on such cases, and possibly have more surgery with removal of these cysts.

Even though the nerve damage cannot be repaired, it seems rather a shame to make this diagnosis only to establish the fact that nothing can be done for the patient. I believe we are prone to draw conclusions on the basis of small numbers of examples of rare conditions because it is difficult for any one person to obtain a large series. In a commonplace lesion, such as carcinoma of the stomach, or colon polyps, or ruptured intervertebral disc, one would not draw conclusions from 10, 12 or 15 cases.

Therefore, I believe we can stimulate our neurosurgeons to have myelograms done on these cases, and continue to explore them surgically, until we have collected evidence from a larger series of cases (how big, I don't know), before we feel that this is a hopeless condition from the standpoint of treatment.

I wish to congratulate Dr. Whiteleather on presenting something which is new in diagnostic roentgenology; and even though it may prove to be hopeless from the standpoint of therapy, it is a condition worth knowing about.

Dr. WHITELEATHER (closing). I intentionally left out differential diagnosis for the sake of brevity. In several thousand myelograms, I have never seen an arachnoid or dural cyst that might have been confused with this deformity. The few that I have seen have been in the lumbar region and none have projected through a foramen. It is conceivable that an extradural tumor might effect a defect such as the blunting of a root sleeve or notch but it would most likely produce signs and symptoms of cord pressure. All of these cases presented the distinct history of trauma and such a clear cut neurological syndrome that the neurological surgeons were frequently able to predict the exact roots involved.

Only when an associated lesion has been present, such as in our Case 11, which had an old extruded disc, has there been any doubt. Pain has not been a prominent feature though one man did have causalgia, the exact cause of which was never determined.

I wish to thank Dr. Peterson for his interesting and excellent discussion.

Vol. 72, No. 6

LEIOMYOMA OF THE STOMACH*

By HERBERT L. ABRAMS, M.D.

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THE melancholy outlook confronting the patient with a malignant gastric neoplasm needs no emphasis. There is a group of benign gastric tumors, on the other hand, in which the surgeon can confidently predict cure following removal, and among these are the leiomyomas, or smooth muscle tumors. In the past, benign tumors misdiagnosed as carcinoma clinically and roentgenographically have been deliberately neglected as inoperable. With careful evaluation of the diagnostic criteria, however, a correct tentative diagnosis may be suggested. This is of further aid to the surgeon in that it may alter his plan of surgical therapy, even among those cases in which exploration is mandatory.

In this paper, the characteristics of the smooth muscle tumors of the stomach will be assayed by evaluating 5 hitherto unreported cases, all proved during life, reviewing the pertinent literature, and analyzing reports of 50 collected cases.† [4–11,13–15,18–24,29–34,34,35]

REPORT OF CASES

CASE I (Fig. 1–5). (Patient of Dr. Edmund Butler.) L. L., a forty-five year old woman, entered the hospital with a chief complaint of epigastric distress of about one month's duration. She had had episodes of dizziness and headaches for the past few years. She also had a gradual onset of increasing anemia for one year prior to entry, for which she had received treatment elsewhere without benefit. She had no history of hematemesis or melena.

On physical examination, she appeared slightly pale, but in no acute distress. There was tenderness in the upper abdomen; no masses or viscera were felt.

Hemoglobin was 11 grams. She had a normal gastric acidity, although there was blood in the gastric washings on all specimens taken.

† These will be referred to throughout as the "collected cases" and include the 5 cases reported herein.

Upper gastrointestinal series showed a large ovoid filling defect in the fundus of the stomach. The mucosa adjacent to the filling defect appeared normal. A large central ulceration was present. No disturbance of gastric mobility or peristalsis was observed. The roentgenologic diagnosis was: "tumor of the stomach, with ulceration."

Course. At exploration, a large ovoid mass was present in the fundus of the stomach. Local excision of the tumor was performed and the patient had an uneventful recovery.

Pathologic Report. The gross specimen consisted of a lobulated tumor, with smooth surface, weighing 135 grams and measuring 8 by 6 by 5.5 cm. There were two ulcer craters, 12 and 8 mm. in size respectively, which penetrated the substance of the tumor for about 2 cm. and communicated with each other beneath a 1 cm. broad bridge of intact mucosa. On histopathologic examination, the tumor was composed of interlacing bundles of smooth muscle cells. The diagnosis was: leiomyoma, stomach.

CASE II (Fig. 6–9). H. F., a sixty-two year old man, entered the hospital with a chief complaint of shortness of breath of three weeks' duration. He also had anorexia, some bloating in the abdomen after eating, and a dull ache in the epigastrium.

On physical examination, he appeared to be in no distress. His blood pressure was 140/90. His heart was slightly enlarged. The lungs were clear. The liver was felt three fingers below the costal margin, but no other abdominal organs or masses were palpable.

The hemoglobin was 14 grams, with a normal white blood cell count.

Upper gastrointestinal series showed a round, sharply demarcated filling defect in the gastric cardia. The surrounding mucosal folds appeared normal. The roentgenologic diagnosis was: "discrete tumor of the gastric cardia, probably carcinoma."

Course. At exploration, a round, firm, sharply demarcated mass was found in the cardia of the stomach. This was excised locally,

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Fig. 1. Case 1. Upper gastrointestinal series. Upright spot film demonstrates a large oval mass projecting into the fundus.

Fig. 2. Case 1. Spot film, showing central ulceration in the mass.

Fig. 3. Case 1. Prone oblique film. The adjacent mucosa appears normal, and the edges of the tumor are sharply delineated.

Fig. 4. Case 1. Cut section of the gross specimen. Note the whorled appearance. The two craters communicated with each other.

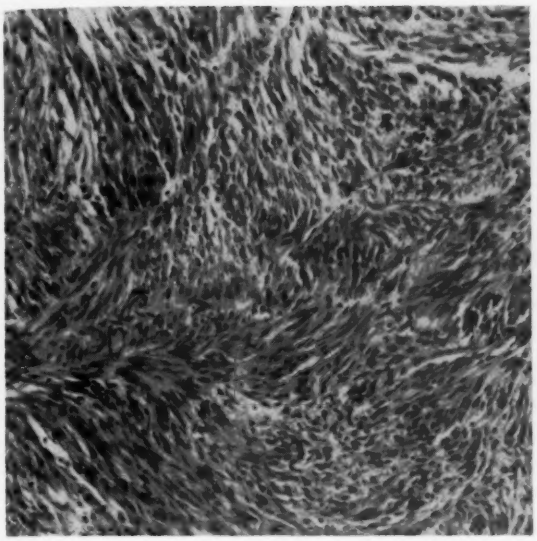

Fig. 5. Case I. Photomicrograph. The nuclei in longitudinal section are oval. The muscle fibers are arranged in curving bands that merge with other groups of muscle fibers.

after frozen section had established its benign nature. The patient did well in the immediate postoperative period. One week after operation,

Fig. 6. Case II. Prone oblique film of the barium filled stomach. There is a possible filling defect in the cardia.

Fig. 7. Case II. Upright spot film. A definite mass impinges on the air filled fundus.

he suddenly became acutely dyspneic and cyanotic and died, apparently having suffered a

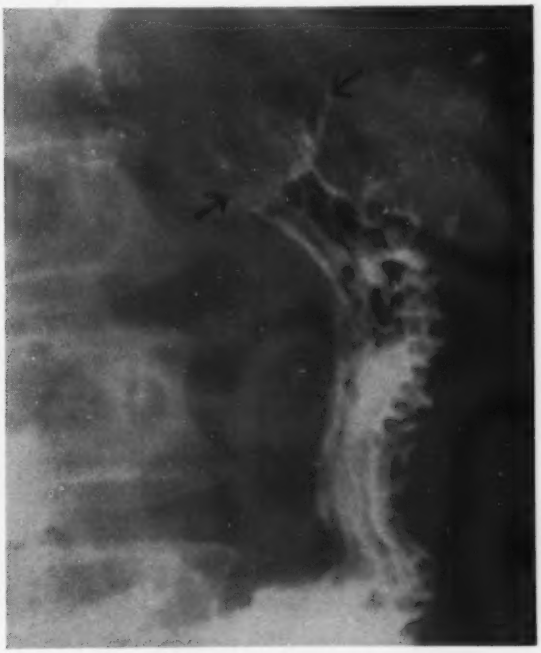

Fig. 8. Case II. Mucosal detail spot film. The rugae are normal adjacent to the tumor and stop abruptly at its edge.

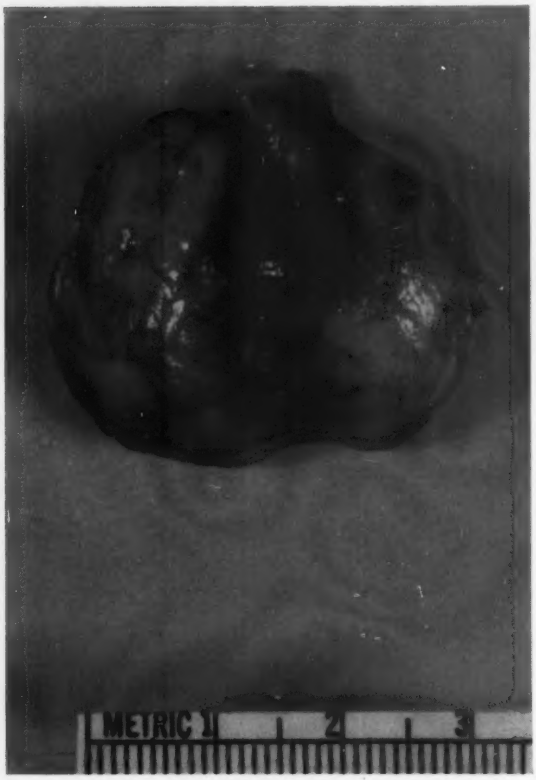

Fig. 9. Case II. Gross specimen. The tumor is slightly lobulated, with a smooth surface.

massive pulmonary infarction. No autopsy was obtained.

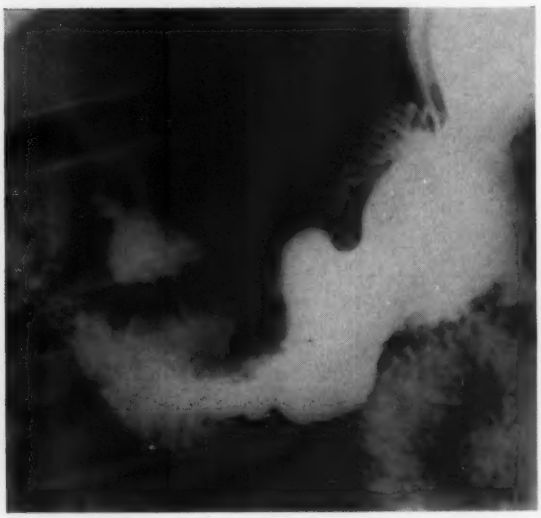

Fig. 10. Case III. Prone oblique film. There is a sharply circumscribed filling defect in the gastric antrum. Peristalsis passed readily through the involved area.

Pathologic Report. The specimen was a 3 by 2 by 2.5 cm. firm round mass, surrounded by a thin connective tissue capsule. On histopathologic examination, irregular whorls of smooth muscle and connective tissue were seen. The cells were uniform, and showed little mitotic activity and no evidence of malignancy. The diagnosis was: leiomyoma, stomach.

Case III (Fig. 10–11). (Patient of Dr. Emile Holman.) G. W., a fifty-six year old man, entered the hospital with a chief complaint of melena. Five weeks before he had become faint, pale and dizzy, and passed a large amount of blood per rectum. He was hospitalized at that time and gradually improved. Two days before admission he again had an attack of melena, became weak and developed marked pallor. He had no nausea or vomiting, nor abdominal pain.

On physical examination, he was a pale but well nourished man in no distress. The heart and lungs were normal. On abdominal examination, no masses or viscera were felt and there was no tenderness.

The hemoglobin was 8 grams. On gastric analysis, there were 23 units of free acid.

Upper gastrointestinal examination showed a discrete mass in the antral portion of the stomach, with a crater in the center. Peristalsis appeared to pass readily through this area, and the mucosal pattern up to the margins of the lesion appeared essentially normal. The roent-

Fig. 11. Case III. Upright spot film shows an apparent niche in the tumor.

genologic diagnosis was: "tumor of the gastric antrum, fibroma or carcinoma."

Course. At operation, a movable mass was felt on the posterior wall of the stomach in the antrum. A subtotal gastric resection was performed. The patient had an uneventful postoperative course.

Pathologic Report. The specimen consisted of an oval portion of the stomach wall, with a large round firm mass 3.5 cm. in diameter in the submucosa. An ulceration in the mucosa about 5 mm. across was present directly over the mass. The remainder of the mucosa appeared essentially normal. The tumor was freely movable, rubbery in consistency, with a smooth contour and a thin delicate capsule. Histopathologic examination showed a pattern of interlacing whorls of long spindle cells with a delicate fibrillary intercellular substance. The diagnosis was: "leiomyoma, stomach, with ulceration."

Case IV (Fig. 12–14). (Patient of Dr. Arthur Bloomfield.) R. B., a fifty year old man, entered the hospital because of a past history of gastric hemorrhage. Twenty years before, he had been operated upon for a duodenal ulcer, a pyloroplasty having been performed. He remained well until about a year before admission, at which time he had two attacks of massive hematemesis. At the time, an upper gastrointestinal series was performed, which was said to show a tumor in the pars media of the stomach. Although the diagnosis of carcinoma was made at that time, the patient refused operation and felt well until the time of admission. He entered the hospital for evaluation of the gastric tumor.

Fig. 12. Case IV. Upper gastrointestinal series. A large, circumscribed filling defect, with central ulceration, is present in the pars media of the stomach on the greater curvature.

Fig. 13. Case IV. Spot film, showing mucosal folds stopping abruptly at the edge of the tumor.

Physical examination was essentially negative.

The hemoglobin was 15 grams and the red blood cell count 5 million.

Upper gastrointestinal series disclosed a discrete mass in the pars media of the stomach, impinging on the greater curvature. Normal mucosal folds were demonstrated adjacent to the mass and the surface of the mass was clearly delineated. There was a niche in the

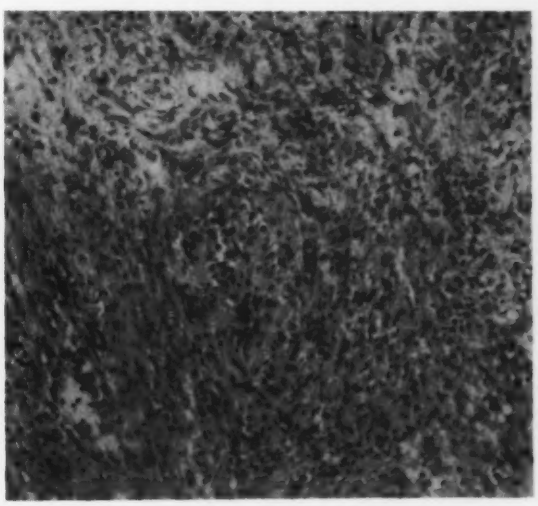

Fig. 14. Case IV. Photomicrograph. In cross section, the smooth muscle nuclei vary considerably in size and shape—because the cells are cut at different levels.

Fig. 15. Case v. Upper gastrointestinal series. A large, discrete filling defect, with a central ulcer crater, is present on the lesser curvature of the stomach, extending almost up to the cardia.

center of the tumor. The roentgenologic diagnosis was: "large submucous tumor, probably benign, with central ulceration."

Course. On exploration, a firm, round, smooth tumor mass was felt, with its base on the posterior wall of the stomach. In the center of its gastric surface was a deep ulcer crater. Local excision was performed and the cut edges of the stomach were approximated. The patient had an uneventful recovery.

Pathologic Findings. The specimen was an ovoid, firm, rubbery tumor, which measured 5 by 3.3 by 4 cm. In the center of the mucosal surface there was a large, deep, penetrating ulcer about 1.5 cm. wide and approximately the same depth. This ulceration extended into the tumor itself. The cut surface was pale and slightly translucent. Histopathologically, the tumor was composed of smooth muscle. The diagnosis was: "leiomyoma, stomach, with ulceration."

Case v (Fig. 15, 16). G. B., a fifty-five year old man, entered the hospital complaining of pallor, weakness and shortness of breath. He had had no nausea, vomiting, abdominal pain, hematemesis, or melena.

On physical examination, he appeared pale and weak. The heart was not enlarged and the lungs were clear. No abdominal masses or organs were felt.

The hemoglobin was 5.5 grams, and the red blood cell count 2.2 million. Stools were markedly positive for occult blood. Gastric analysis disclosed a free acid of 10–15 units, and a total acid of 55 units.

Upper gastrointestinal series showed a large, discrete filling defect on the lesser curvature of the stomach, extending almost up to the cardia. In the center of this mass a deep niche was demonstrated. The adjacent mucosa appeared essentially normal and there was no evidence of infiltration beyond the limit of the circumscribed mass. The diagnosis was: "large gastric tumor with ulcer crater."

Course. The patient was prepared for surgery by repeated transfusions of whole blood. At operation, a large, well circumscribed tumor mass was found on the lesser curvature in the anterior wall of the pars media. The tumor was approximately 5 cm. in diameter, and projected into the lumen of the stomach for a distance of about 4 cm. In its center was a large deep ulcer crater. A subtotal gastrectomy was

Fig. 16. Case v. Operative specimen. The tumor is smooth and sharply circumscribed, with a deep ulcer crater. The adjacent mucosa appears normal.

performed, and the patient had an uneventful recovery.

Pathologic Report. The specimen consisted of a portion of stomach measuring 18 by 11 cm. On the lesser curvature there was a discrete globoid mass about 6 cm. in diameter, extending into the lumen. The mucous membrane was stretched thin and smooth over its surface. There was a 2 cm. crater at the top of the dome, extending 1 cm. into the depth of the tumor. Histopathologically, the tumor was composed of spindle-shaped cells arranged in bundles, with the typical appearance of smooth muscle. The diagnosis was: "leiomyoma, stomach, with ulceration."

DISCUSSION

1. *Incidence.* Until recent years, benign tumors of the stomach were considered a rarity.[12,17] In 1936, however, Rigler and Ericksen[31] reported that 24 per cent of a large group of gastric tumors noted pathologically were benign, and that 11 per cent of gastric tumors found on roentgen examination were benign. Support for this higher incidence of benign gastric tumors was furnished by Dudley and his coworkers,[10] who found that 22 per cent of all gastric tumors discovered in an autopsy series were benign, although only 1.3 per cent of their surgical series were benign.

Of the benign gastric neoplasms, leiomyoma is certainly the most common type.[2,6,8,25,27] Rigler and Ericksen found that leiomyomas comprised 27 per cent of all benign gastric tumors, and Collins and Collins[6] found a 40 per cent incidence among 1,347 reported benign gastric neoplasms. Judd and Hoerner[16] reported that 70 per cent of 50 benign neoplasms found at operation were leiomyomas. Rieniets,[30] in a pathologic study of 200 consecutive autopsied cases, found an incidence of 13 per cent of gastric leiomyomas. Meissner[26] found leiomyomas in 23 of 50 stomachs carefully studied histopathologically, an incidence of 46 per cent.

One explanation for the varying reports and for the discrepancy between autopsy and surgical figures is that most leiomyomas found at autopsy are small, and average about 0.5 cm. in size. Usually, they cause no symptoms, and hence fail to attract attention during life. During the past fifteen years, 50 instances of gastric leiomyoma are recorded in the pathological files of the Stanford University School of Medicine, but of these only 5 were of sufficient clinical importance to warrant operation.

2. *Age and Sex.* The overwhelming majority of gastric leiomyomas are found between the ages of forty and sixty.[4,7,14,25] In the collected cases, only 1 was below the age of forty, and 70 per cent of the patients were in the fifth and sixth decades of life.

No predilection for either sex was apparent.[14,21,25] Twenty-six of the collected cases were male, and 24 female.

3. *Pathology.* These tumors originate in the muscular layer of the stomach, and although most are submucosal and intragastric in location, occasionally they are extragastric or subserosal.[5] The smaller ones may remain in the wall as an intramural lesion, without projecting either toward the peritoneal cavity or toward the lumen of the stomach.[25]

When found at autopsy, they are usually below 1 cm. in size.[26] They may attain enormous size, however, in spite of their apparent slow rate of growth (Fig. 4). Grossly, they are firm, well defined, sharply circumscribed tumors.[14] The smaller growths are usually ovoid or round, and the larger ones may be lobulated.[25] The cut surface is grayish-pink or cream colored, with a whorled appearance. Because they are relatively avascular, degenerative changes such as fibrosis, hemorrhage, hyaline degeneration and calcification are common.[14] Ulceration occurs frequently and tends to be quite deep (Fig. 4 and 16). Figures 4, 9 and 16 display the characteristic gross appearance. In Figure 16, the normal adjacent mucosa, clear-cut borders, smooth surface and deep ulceration are well demonstrated.

On histopathologic examination, interlacing bundles of smooth muscle fibers are

seen (Fig. 5). The cells are spindle shaped, well differentiated and show rare mitoses. Occasionally, palisading is present, which at times causes confusion with nerve sheath tumors. The nuclei are cigar shaped and slightly more pleomorphic than normal smooth muscle. In cross section, with the cells cut at different levels, there may be an apparent marked difference in size and shape (Fig. 14).

At times a pointless distinction is made in the literature between gastric fibromyoma and leiomyoma. The only difference between the two is the relative amount of connective tissue stroma, which probably multiplies as the tumor increases in age and size.[23] With a hematoxylin and eosin preparation, the muscle takes a deeper and more purple stain than connective tissue, and is not as refractive.

It may be difficult to differentiate between a benign leiomyoma and a leiomyosarcoma,[14] and there have been reports of supposedly benign leiomyomas metastasizing to the liver. In these cases, mitotic figures, pleomorphism, and evidence of invasion have been absent.

4. *Location of the Tumor.* Many of the articles in the literature emphasize that an overwhelming number of tumors are located in the pyloric region.[1,4,13,14,20,25] Others have reported a higher incidence in the cardia,[5,6] while a third group has found that there is no predilection for any special part of the stomach.[27] Collins and Collins[6] pointed out that 85 per cent of postmortem specimens were situated at the cardia, while 65 per cent of the surgical specimens were situated near the pylorus. These figures caused Covey and Rogers[8] to suggest that for some reason leiomyomas in the distal third of the stomach tend to grow to greater size and to cause more symptoms than elsewhere. In the collected cases, the distribution of tumors between the pyloric end and the body of the stomach was roughly equal, with a somewhat smaller number in the fundus and the cardia. More were located on the greater curvature than on the lesser curvature. The variable location is well demonstrated in the roentgenographic illustrations.

TABLE I

INCIDENCE OF SYMPTOMS AMONG 50 COLLECTED CASES OF LEIOMYOMA

	Number of Cases	Percentage
Bleeding	33	66
Manifestation		
Hematemesis	10	
Melena	13	
Hematemesis and melena	5	
Unspecified	5	
Associated Symptoms		
None	15	
Epigastric distress, pain, "dyspepsia"	13	
Epigastric pain and distress	9	18
Obstruction, nausea and vomiting	4	8
Anorexia and weight loss	2	4
Slowly enlarging abdominal mass	1	
No symptoms	1	

5. *Symptoms and Physical Findings* (Table I). Hemorrhage is by far the commonest symptom,[1,3,4,14,21,25] and was present in 66 per cent of the collected cases. Recurrent attacks were frequently noted. In about one-half of the cases with bleeding, a history of abdominal distress of relatively long duration was obtained. In 4 of our 5 patients, bleeding was the primary manifestation of the tumor.

Epigastric pain and weight loss,[13] a peptic ulcer syndrome,[5] pyloric obstruction,[14] and a palpable mass were also noted in a significant number of cases.

The duration of symptoms was stated in 32 of the collected cases, in 24 of which it exceeded one year (Table II).

6. *Laboratory Findings.* Although it has been stated that achylia is present in most cases,[13] it seems apparent that there is no constant relationship to gastric acidity.[4,20] Among 17 patients whose gastric acidity was tested, 10 had no free acid and 7 had normal or high acidity.

The stool guaiac test was positive in the majority of patients on whom the examination was performed. The hemoglobin

Table II

DURATION OF SYMPTOMS IN 32 CASES OF GASTRIC LEIOMYOMA

Duration	Number of Cases
1 month or less	5
2 to 12 months	3
1 to 2 years	8
2 to 5 years	6
5 to 10 years	3
10 to 25 years	3
Many years	4

was recorded in 22 instances, in two-thirds of which it was below 10 grams.

7. *Gastroscopy.* On gastroscopic examination, the leiomyoma is seen as a smooth, sharply limited tumor, with the overlying folds stretched and effaced. There is no evidence of induration surrounding the tumor site. Schindler *et al.*[33] have described as a sign of a submucosal neoplasm the stretching of a mucosal fold from the surrounding mucosa up to the surface of the tumor, bridging the space between the top of the tumor and the level of the surrounding mucosa. In at least 1 case in which gastroscopy was performed, this sign was negative.

8. *Roentgenologic Findings.* In 1924, Moore[28] elaborated a set of criteria for the diagnosis of benign gastric tumors to which relatively little has been added. These tumors produce a circumscribed filling defect, over which the rugae are obliterated. The surrounding folds, however, are normal, and there is no disturbance in peristalsis. Moore stated at the time, and this was later emphasized by Carter and Laing,[3] that benign tumors do not reveal a niche; this statement certainly does not apply to the smooth muscle tumors.

Leiomyomas are usually round or oval and sharply demarcated.[31] The margins are smooth, and the tumor is mobile on palpation under the roentgenoscope.[4] There is no evidence of fixation or induration,[13] and the stomach wall is pliant.[14] Although they are occasionally pedunculated, more frequently they are sessile. The filling defect frequently forms an abrupt angle with the uninvolved gastric wall, which helps to distinguish it from an extrinsic pressure defect.[32] Ulceration is common,[13,16,25] but in the presence of ulceration, it is difficult to demonstrate folds converging toward the center of the crater. The ulcers are usually deeper than the ordinary simple peptic ulceration.

In the over-filled stomach, barium may obscure the location of the tumor,[31] and under these circumstances, compression is helpful. Heavy penetration may also bring out the filling defect.

Among the 50 collected cases, upper gastrointestinal examinations were performed in 41, 2 of which were negative, and in the remainder of which a definite mass was demonstrable. In 28 of these cases, there was ulceration of the leiomyoma. The filling defect was almost invariably sharp and circumscribed, and the surrounding mucosa appeared essentially normal. In 2 instances, calcification of the tumor was demonstrable on the plain film. I have personally seen 1 case of a gastric leiomyoma, about 1 cm. in size, which could be seen on the plain film as a calcific density in the left upper quadrant.

In Figure 1, the large soft tissue mass is clearly outlined. Its smooth surface and central ulceration, as well as the adjacent normal mucosal folds, are well demonstrated in Figures 2 and 3. The clearcut filling defect of the cardia shown in Case 11 was constant at roentgenoscopy (Fig. 7). Figure 11 is a pressure spot film which brought out the niche in Case 111. The typical, sharply delineated filling defect, with a deep ulcer crater, is shown in Figure 15.

It is important, although difficult, to assess the reliability of the criteria for diagnosis. Most of the cases described in the literature were diagnosed preoperatively as carcinoma. "Gastric tumor" was usually the diagnosis in our cases, in 2 of which a benign lesion was suggested. It would seem, then, that these criteria have not been too useful in the past.

But what has actually happened is that

the radiologist, swayed by the statistics, has tended to make the diagnosis of carcinoma in all cases of gastric tumor, because it is the commonest tumor discovered roentgenologically. In addition, if a gastric carcinoma proves to be a benign neoplasm, the error in diagnosis is seldom the cause of uproar by his colleagues that the reverse situation provokes. Yet, painstaking efforts at differential diagnosis may prove to be of great value to the surgeon.

I think it is possible to reach a middle ground in which any tumor which meets the criteria described above can be called "probably benign." If, in addition, it appears to be intramural in location and presents a single deep ulcer, the possibility of leiomyoma may be suggested. Although there are other intramural tumors, a leiomyoma is certainly the commonest and the one which most frequently ulcerates. When the diagnosis of a benign tumor is made, it should be made clear to the referring physician that the possibility of malignant neoplasm cannot be entirely excluded.

9. *Treatment.* Radical gastric resection has been advocated as the treatment of choice because of the "frequency" of malignant degeneration and because benignity is difficult to establish at the time of operation, even with a frozen section.[20] Others feel that if the tumor is benign and localized, local excision is acceptable.[14] This general schism in attitude is reflected in most of the literature. On the whole, however, by far the greatest number of tumors have been locally excised, and there has been very little evidence of recurrence. In 3 of our patients, local excision was performed, and in 2, subtotal gastric resection.

Exploration is mandatory, unless specifically contraindicated, regardless of how strongly the roentgenograms and gastroscopy may indicate benignity. Until the tumor is examined histopathologically, the presence of a malignant lesion cannot be totally excluded. In addition, if the tumor is large enough to be roentgenographically demonstrable, the chances are reasonably good that bleeding episodes will occur sooner or later. In 5 of the 50 collected cases, surgery was not employed; 4 died because of bleeding from their leiomyomas, and 1 died following an unrelated illness.

10. *Prognosis.* The prognosis in leiomyoma of the stomach is excellent. Among the cases subjected to surgery, there have been very few reported recurrences. In spite of the high rate of malignancy which would seem to be indicated by Lahey and Colcock,[20] the majority of the reports in the literature support the point of view that these lesions rarely degenerate into leiomyosarcoma. Most deaths have been caused by the effects of ulceration of the tumor. Conservative treatment, as noted above, is not indicated. In Thompson and Oyster's[34] series of 7 patients, the 3 conservatively treated all died. All of our patients survived the operation, 1 dying of pulmonary embolism a week later. There has been no evidence of recurrence in the remaining 4 cases.

SUMMARY

1. Five cases of leiomyoma of the stomach discovered at operation are presented, with the pertinent clinical, pathological and roentgenological findings.

2. The literature is reviewed and the diagnostic criteria are summarized and evaluated.

3. The demonstration of a smooth, round, sharply demarcated, sessile gastric tumor, with central ulceration, normal adjacent mucosa, and normal peristalsis should suggest the possibility of a leiomyoma of the stomach.

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RETROPNEUMOPERITONEUM ASSOCIATED WITH LAMINAGRAPHY*

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RETROPERITONEUM is a procedure for the visualization of the retroperitoneal organs by means of an artificial emphysema of the retroperitoneal tissues. The method may be supplemented by insufflation of hollow viscera, by intravenous urography and by other procedures. Retroperitoneum in combination with laminagraphy has been very useful to us in the study of the kidneys, the pancreas and the adrenals.

In most instances, we have found the procedure to be of easy execution. The insufflation, however, does not succeed when old inflammatory processes have caused fibrosis of the retroperitoneal fatty tissues. The method is contraindicated in the presence of active retroperitoneal inflammation and also in cases of capillary fragility and defective coagulation of blood.

TECHNIQUE

We prepare the patients with cleansing enemas, then sterilize the skin of the perianal region. With the patients in the genupectoral position we introduce a needle in the midline, 1 cm. in front of the tip of the coccyx, in an oblique posterior direction, until the needle finds the anterior wall of the coccyx at a depth of about 4 cm. The

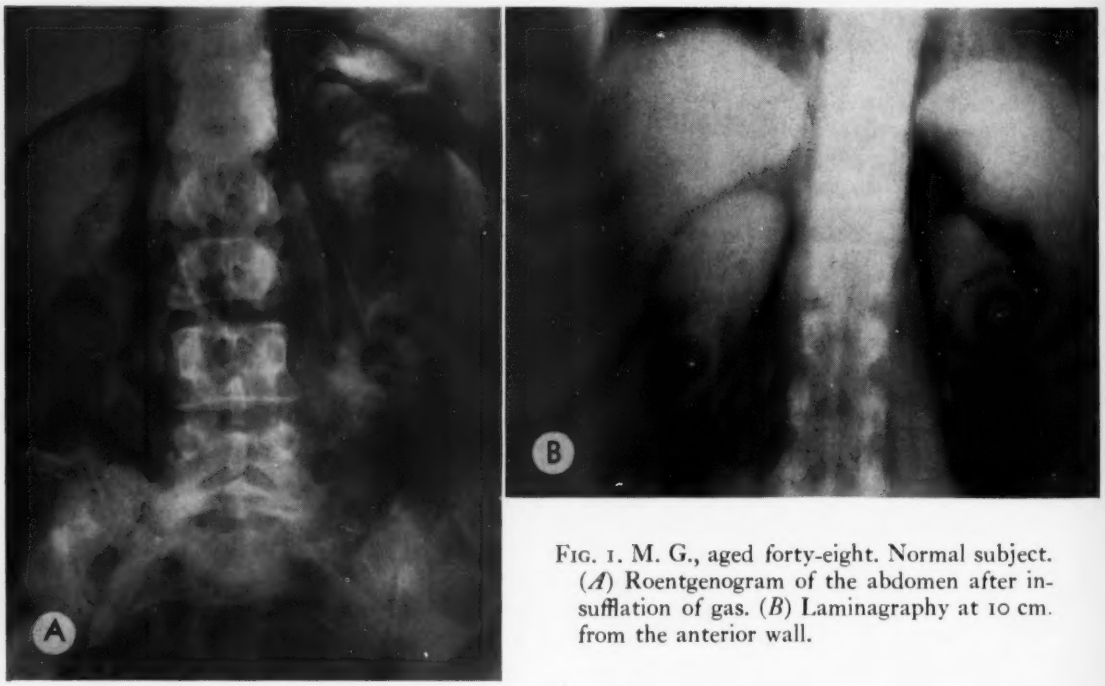

FIG. 1. M. G., aged forty-eight. Normal subject. (*A*) Roentgenogram of the abdomen after insufflation of gas. (*B*) Laminagraphy at 10 cm. from the anterior wall.

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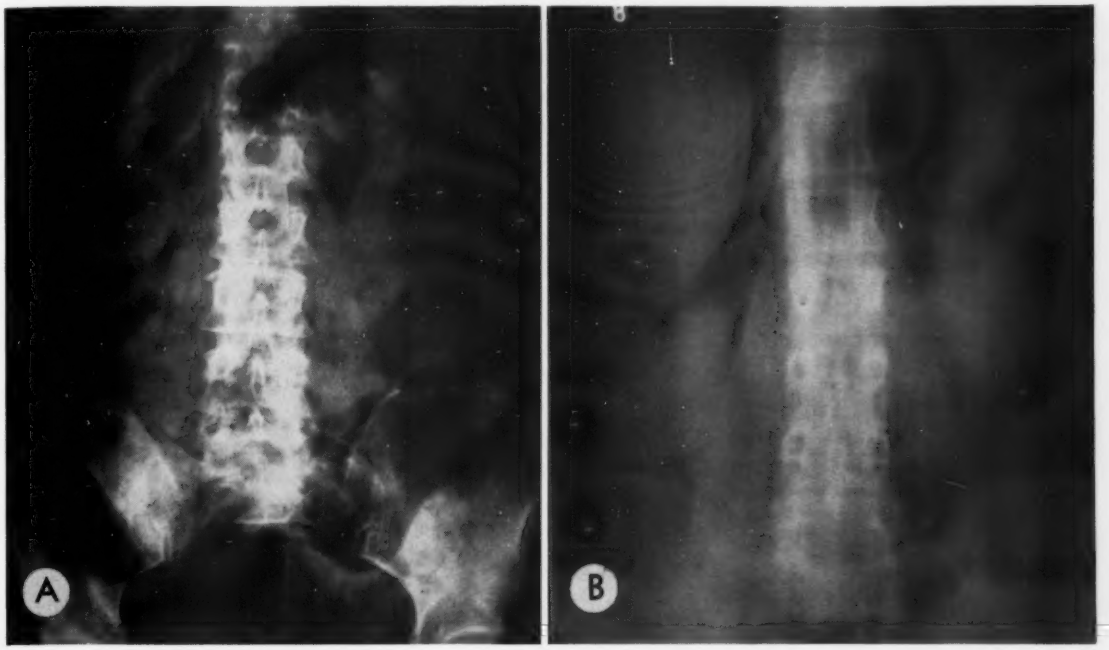

Fig. 2. B.A., aged sixty-four. Hepatomegaly and right renal ptosis. (*A*) Roentgenogram of the abdomen after insufflation of gas. (*B*) Laminagraphy at 8 cm. from the anterior wall.

injection must be made above the ano-coccygeal ligament and for this reason, it is best to establish contact with the bone at about 4 cm. of depth. When the needle is in

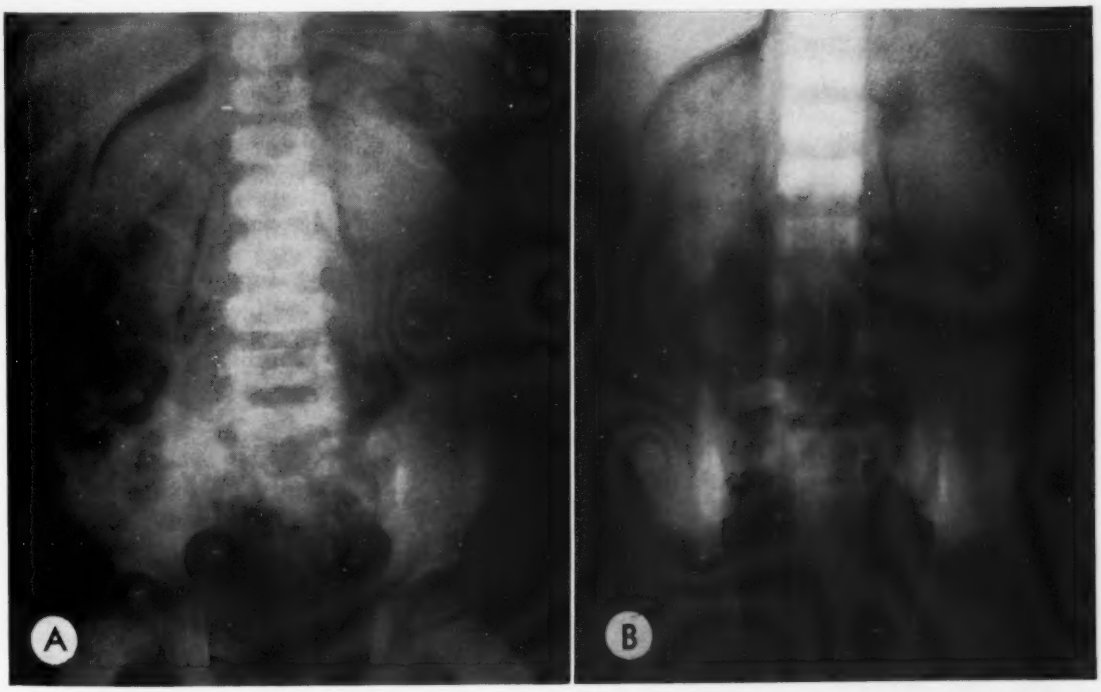

Fig. 3. M.P., aged three. Hypernephroma of the left kidney, confirmed by operation. (*A*) Roentgenogram of the abdomen after insufflation of gas. (*B*) Laminagraphy at 10 cm. from the anterior wall.

position and if blood cannot be aspirated, we introduce 500 cc. of air or oxygen in children, and 1,200–1,500 cc. of gas in adults. The gas will cause a tympanitic sound upon percussion of the lumbar areas. During insufflation the patients show no marked discomfort, but a sense of painful fullness may be experienced for a few minutes, first in the left, then in the right lower quadrant. The patients are asked to sit up for about thirty minutes, and then they are roentgenographed in the supine position. The direct roentgenographic examination is quite useful in most cases, but even better definition may be obtained by frontal and lateral laminagrams aimed at various body depths. The frontal laminagrams are especially useful for the study of the kidneys. Lateral laminagrams are utilized in conjunction with gastric insufflation for the study of the pancreas and also for the demonstration of tumors of the gastric fundus. The Trendelenburg position allows a good visualization of the female pelvic organs.

We have employed pneumoperitoneum in over 276 patients. No accidents have occurred in our experience with the method with the exception of a retroperitoneal hemorrhage in a patient with defective blood clotting. This could have been avoided if appropriate blood studies had preceded the examination. Perforation of the rectal wall by the needle has been reported. This is not particularly dangerous and may be promptly detected because of the tenesmus which it causes. When the needle is too superficial, the injection of gas will produce emphysema of the perineal tissues. This is easily seen and the needle should be re-inserted more deeply.

In our experience, the retroperitoneal insufflation of gas is a very useful procedure which seems to be without essential dangers. We believe that the simplicity of the method makes it preferable to the perirenal injection of gas for the study of the kidneys and of the adrenals.

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ANEURYSM OF THE HEPATIC ARTERY DEMONSTRATED ROENTGENOGRAPHICALLY*

A CASE REPORT

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ANEURYSMS of the hepatic artery are relatively rare lesions. Whereas 88 have been reported, their roentgen manifestations seem to have been entirely overlooked. This communication will review briefly the literature dealing with hepatic artery aneurysms. It will also record our roentgen findings in a patient suspected of having such an aneurysm which was proved at postmortem examination.

INCIDENCE

Aneurysms of the hepatic artery occur much more commonly in men than in women (62:20).[3] Preferring no age group, they have been reported in a child ten years of age and in a man of eighty.

ETIOLOGY

Malloy and Jason[3] considered them the result of infection in 60 per cent, arteriosclerosis in 22 per cent, and trauma in 10 per cent of the cases. Syphilis, common cause for aneurysms in the large arteries, was not a prominent etiologic factor in this group, occurring in but 6 individuals. Five patients had associated cholelithiasis. Malloy and Jason felt that in the latter the aneurysms were due to cholangitis rather than vascular pressure by the concretion.

PATHOLOGY[1,2,4,6]

Aneurysms of the hepatic artery may be divided into two large groups: the extrahepatic and the intrahepatic. In the series reported by Malloy and Jason, 61 occurred outside of the liver, 20 within the liver, and 3 aneurysms were found both within and without the liver. Whereas in most instances the aneurysms were single, as many as 4 and 5 were recorded in one individual.

The main trunk of the hepatic artery was most commonly affected, being involved 40 times. Its right and left branches were involved but 26 and 7 times respectively. Noteworthy was the fact that the extrahepatic sacs usually were larger than the intrahepatic ones.

The literature indicated that the smaller hepatic artery aneurysms usually were true aneurysms resulting from dilatation of the vascular wall. The larger ones more often were false aneurysms, or hematomas resulting from rupture of the weakened vascular wall. Both types adhered to surrounding structures producing dense fibrous adhesions which compressed the bile ducts.

Hepatic artery aneurysms, when they burst, usually ruptured into the abdominal cavity or adjacent structures. Of the 85 patients reported by Malloy and Jason, ruptures occurred in 67. In 35 the aneurysms ruptured into the abdominal cavity; in 22 into the extrahepatic bile ducts; in 7 into the duodenum; in 6 into the gallbladder; in 1 into the portal vein; and in 1 the aneurysm ruptured into the stomach.

Arteriosclerotic changes with significant vascular calcification were found in but one-fourth of the entire series of hepatic artery aneurysms. The significance of this observation is obvious. The latter may prove to be the only aneurysms demonstrable roentgenographically.

SYMPTOMS

Pain, hemorrhage and jaundice were the outstanding clinical findings. The pain, which usually occurred in the right upper

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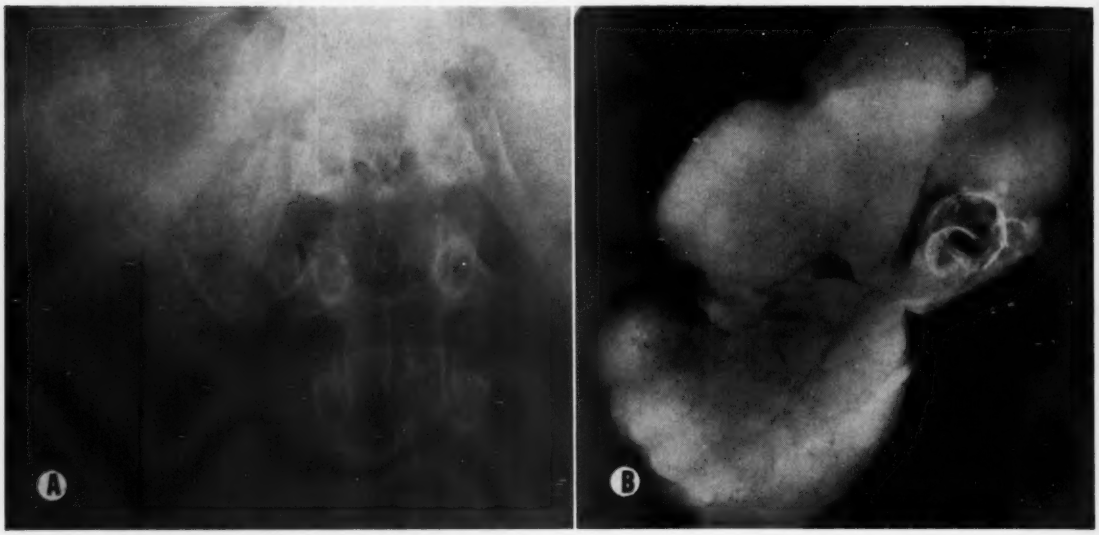

FIG. 1. (*A*) Roentgenogram reveals the hepatic artery aneurysm in the paravertebral region adjoining L1. (*B*) Roentgenogram of the liver at autopsy, which shows the multiloculated aneurysms to better advantage.

abdomen or epigastrium, was described in 58 of the 85 patients reported by Malloy and Jason. Hemorrhage, which occurred in about one-half of the patients, was manifested by profuse hematemesis, melena, or occult blood in the stools. Jaundice, present almost as frequently as hemorrhage, was usually obstructive and the result of compression of the common duct by the aneurysmal sac or blood clot. In many instances the jaundice was intermittent.

Fever, asthenia, constipation, ascites, and an enlarged pulsating liver were also observed.

ROENTGEN MANIFESTATIONS

In spite of the fact that our review of the literature failed to reveal a description of the roentgen manifestations of hepatic artery aneurysms, we feel reasonably certain others must have recognized and described it before us.

The lesion in our patient looked so much like a splenic artery aneurysm roentgenographically except for its position, it seems hard to believe others would have overlooked it.

In our patient the lesion was observed in the survey film of the abdomen made in the anteroposterior projection. It presented itself as a somewhat multiloculated ovoid calcific density in the right upper abdomen. Lying closer to the spine than most gallbladders, one might have confused it with calcification in the wall of the gallbladder. The calcific densities were globular and sharply defined, the periphery being egg-shell in character, the center less dense and mottled. As indicated above, our attention was directed to it because it mimicked aneurysms of the splenic artery so closely. Seids and Hauser's[5] description of the latter suited this perfectly.

Not enough aneurysms of the hepatic artery have been described roentgenographically to permit one to establish criteria for its roentgen diagnosis. Obviously, its recognition will depend upon the presence of calcific debris within the wall of the aneurysm, or perhaps even within the hemorrhagic mass. The fact that but one-fourth of all hepatic aneurysms reveal significant atherosclerosis pathologically suggests that most hepatic artery aneurysms will remain unrecognized roentgenographically.

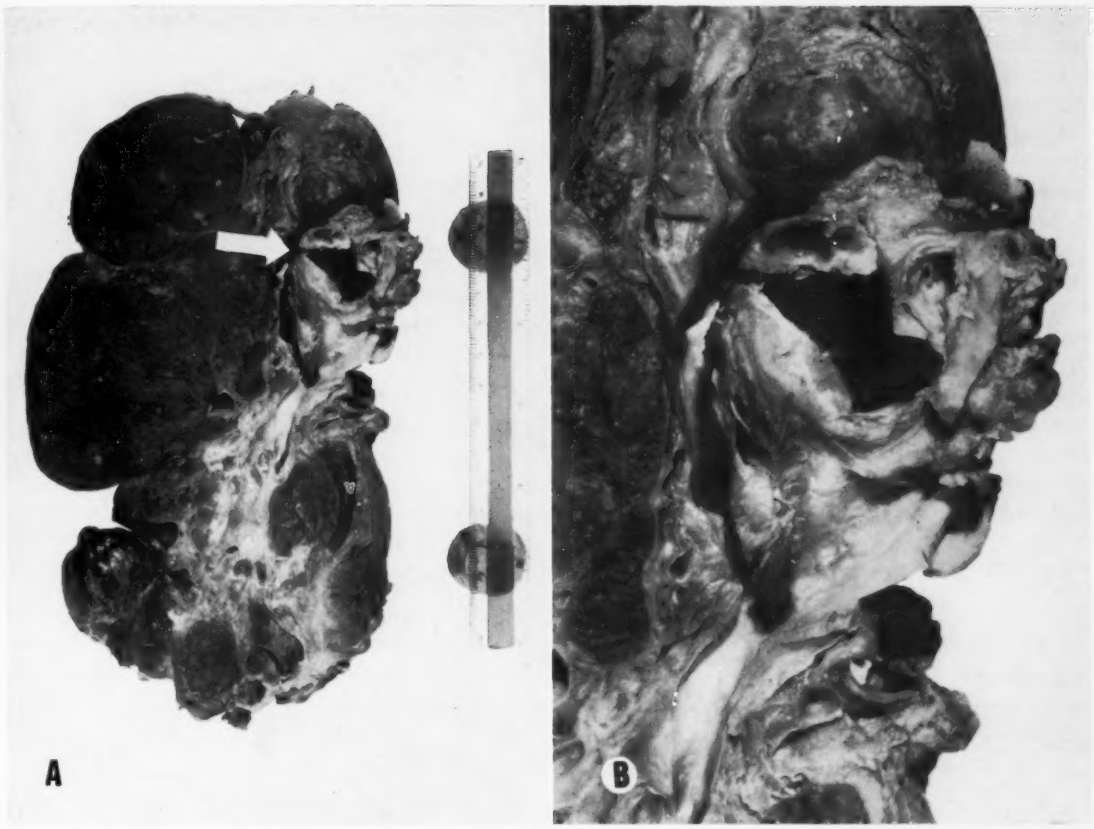

Fig. 2. (*A*) Cross section of the liver revealing multiple tumor nodules. The arrow points to the aneurysm. (*B*) The aneurysm is seen to better advantage in this close-up photograph.

REPORT OF CASE

C.M.B., a colored male, aged seventy-four, was first seen in the Hospital of the University of Pennsylvania in November, 1948, complaining of frequency, nocturia, dribbling and hematuria. At that time he was found to have hepatomegaly with ascites and impaired liver function, all of which was attributed to Laennec's cirrhosis. At that time the patient developed an incomplete small intestinal obstruction for which he was operated upon and a Lane's kink removed. At operation the liver was found studded with small nodules which revealed the presence of hepatic cirrhosis microscopically.

In January, 1949, the patient was readmitted because of ascites. At that time his liver function tests were abnormal. With conservative treatment the ascites subsided and the liver function tests improved.

In October, 1950, the patient's abdomen again began to enlarge and cause discomfort. He complained of dull, persisting generalized abdominal pain which was most severe in the lower abdomen and unrelated to food.

Physical Examination. The significant physical findings were related to the abdomen where the surface veins were observed to be dilated and filling from below. Obvious ascites was present; the liver edge extended 5 cm. below the costal margin.

Laboratory Findings. The patient was moderately anemic with 3 million red blood cells and 50 per cent hemoglobin. The white blood cell count was essentially normal, with 73 to 85 per cent neutrophils. On two occasions urine examination revealed 2 plus albuminuria with many white blood cells. The patient's serology was negative. The blood urea nitrogen ranged from 17 to 50, depending upon the patient's state of hydration. The alkaline phosphatase revealed a slight increase, 15.3 units. The liver flocculation tests were essentially normal.

The patient became progressively weaker and finally died of a massive hemorrhage, presumably from esophageal varices.

Autopsy Findings. Of primary interest was the patient's liver which weighed 2,080 grams. It was entirely distorted by multiple large, irregular nodules, many of which seemed hemorrhagic on the surface. In the region of the hepatic hilum, close to the entrance of the portal vein, was noted a calcified cystic structure intimately related to the hepatic artery, which proved to be an aneurysm of the hepatic artery that measured 3 by 3 cm. (Fig. 2). Distributed throughout the liver were multiple large nodules, some measuring 8 to 10 cm. in diameter, which appeared to occupy 80 per cent of the liver parenchyma.

Histopathologic examination confirmed the presence of the aneurysm. The liver nodules were hepatomas.

SUMMARY

1. Aneurysms of the hepatic artery, although rare, can occasionally be recognized in survey films of the abdomen.

2. Roentgenographically hepatic artery aneurysms look like aneurysms of the renal artery except for their location.

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METHYLCELLULOSE-BARIUM MIXTURE STUDY OF THE COLON

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FOR several months we have added methylcellulose, in the form of collogel,* to our usual barium enema mixture to prevent precipitation of the barium on the colon wall. This has generally made it possible to do a satisfactory double contrast study of the colon immediately following the usual barium enema.

It is accepted that the double contrast study is the method of preference for the demonstration of polyps. Sometimes it also adds to the visualization of other lesions which were first demonstrated by the barium enema. Attempts to produce a double contrast study immediately after the routine barium enema are usually unsatisfactory because of precipitation of the barium on the colon wall, producing a "flaking" effect. As there is reluctance to totally discard the barium enema in favor of the double contrast study, the usual method is to do the two examinations on two separate days, if both are desired. As this means an extra examination, the double contrast study is probably not used as often as it could be to good advantage. Many excellent methods of double contrast study have been presented in the literature. Most, however, are based upon a separate examination for the double contrast study. It is felt that a method which would allow a satisfactory double contrast study to be done immediately after the usual barium enema would be worth while. As this allows the two examinations to be done at a single visit, the addition of the double contrast study becomes a practical one.

The use of methylcellulose with the barium mixture has previously been reported but not emphasized in the radiological literature. Methylcellulose is a synthetic substance made by treating cellulose with methyl chloride, forming an inert hydrophilic colloid. It acts as a gel, preventing drying and precipitation of the barium on the colon wall. We have used it in the form of collogel, which is a commercial laxative preparation containing methylcellulose. The barium enema mixture is prepared as usual in the malted milk mixer and then one level teaspoonful of collogel is added to each mixer canful and allowed to mix for an additional five minutes. The colon is filled with this mixture by gravity as it would be for the usual barium enema. When the head of the column reaches the hepatic flexure, the flow is slowed so that reflux into the terminal ileum is minimized. Roentgenograms are taken and are no different from those of the routine barium enema study. The patient is then allowed three minutes for evacuation and then re-examined by roentgenoscopic study. If it appears that the residual barium is distributed evenly, an evacuation film is taken. Immediately following this, under roentgenoscopy, the colon is insufflated with air. Experience has shown that if the colon wall is adequately coated for a good double contrast effect there will almost always be a sufficient amount of barium in the colon to collect in the more dependent areas, obscuring them. For this reason, roentgenograms are taken in both the anteroposterior and posteroanterior positions so that those areas which are obscured in the one position will be visualized in the other. This method is illustrated by Case 1 (Fig. 1).

When the patient is roentgenoscoped after evacuation, it is more commonly found that the residual barium is distributed unevenly. When this is the case, the patient is allowed an additional twenty minutes to evacuate. By then almost the entire mix-

* We wish to express our thanks to Eli Lilly Company of Indianapolis for supplying the collogel.

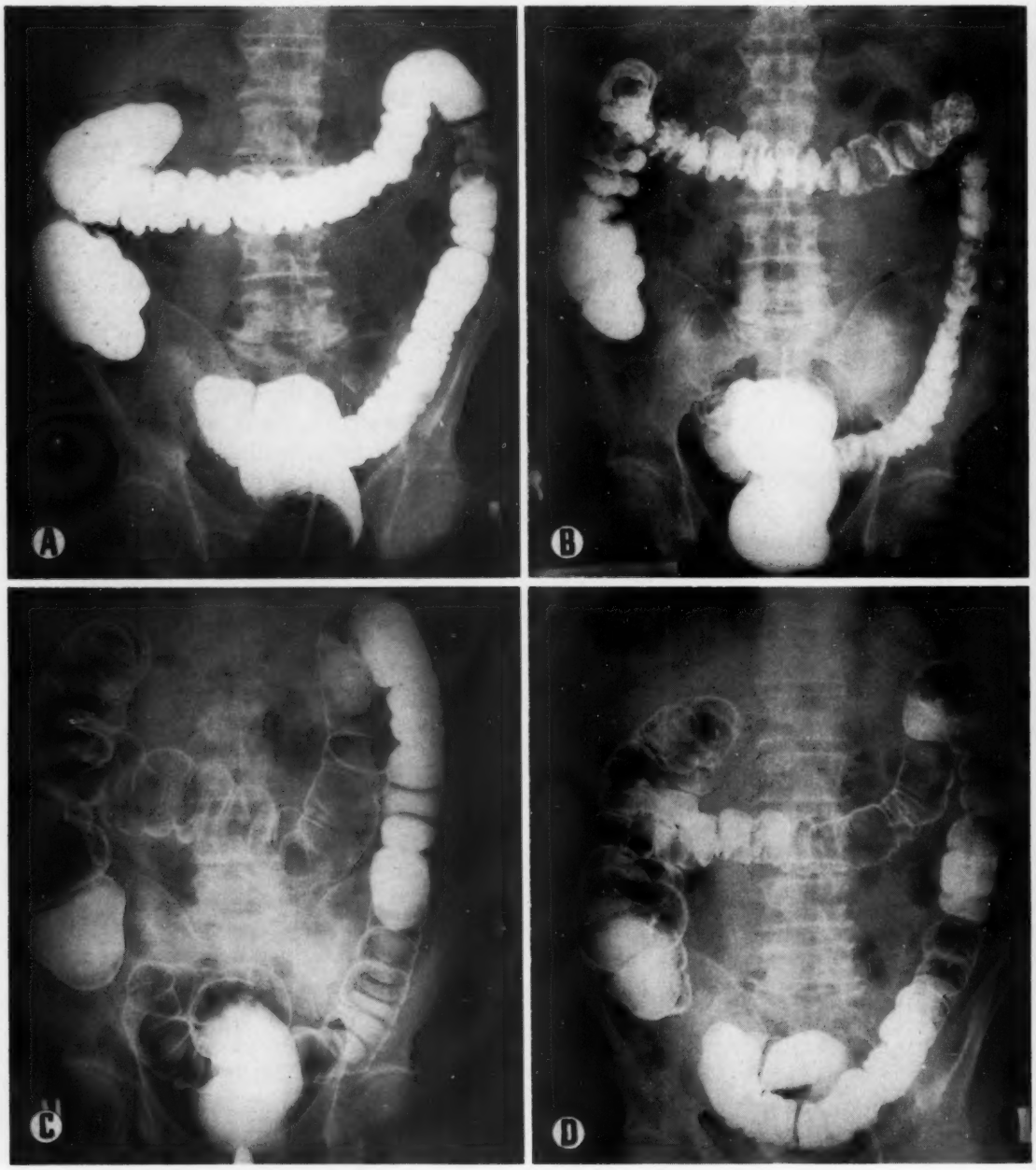

Fig. 1. Case 1. (*A*) Colon filled with barium-collogel mixture. (*B*) Evacuation film after three minutes. (*C*) Posteroanterior and (*D*) anteroposterior views after air insufflation.

ture has been evacuated and the evacuation film is taken. Then the colon is filled to the splenic flexure with the same mixture and by rotation of the patient plus intermittent insufflation with small amounts of air the barium is distributed throughout the colon. Once adequately distributed, the colon is completely insufflated with air. The anteroposterior and posteroanterior views are taken. This method is illustrated by Case II (Fig. 2). Here, after three minutes for evacuation, it was felt after roentgenoscopic study that the distribution of the mixture was poor. There was an excessive

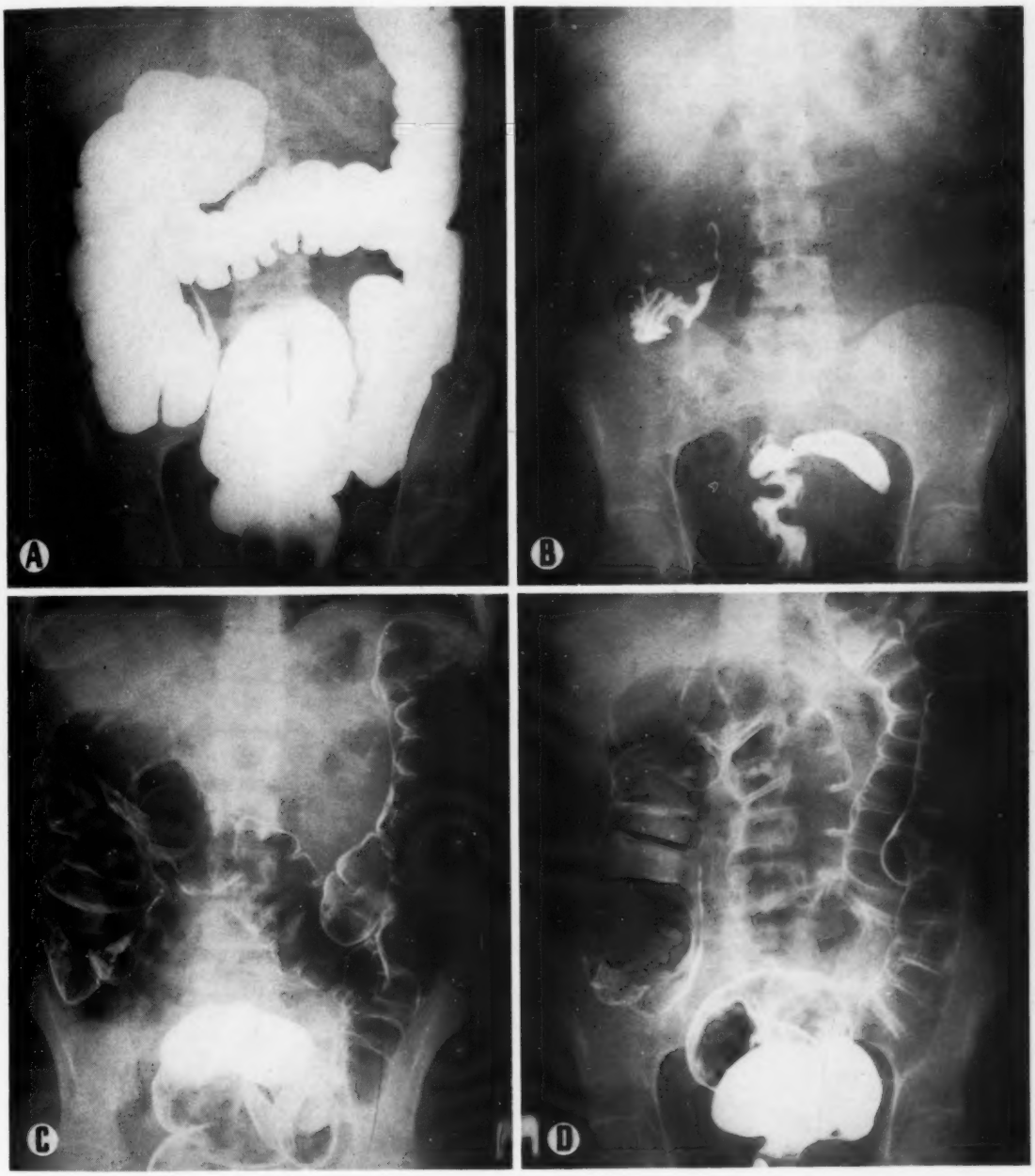

Fig. 2. Case II. (*A*) Colon filled with barium-collogel mixture. (*B*) Evacuation film after twenty minutes. (*C*) Posteroanterior and (*D*) anteroposterior views taken after additional mixture was distributed and then air insufflated.

amount in the cecum and almost none in the descending colon. The postevacuation film shows how complete the evacuation was after an additional twenty minutes. The double contrast films show an absence of precipitation after this additional period.

The use of collogel added to the barium mixture can be done routinely, if desired, as there is almost no additional effort or expense required in its preparation. Even as late as twenty minutes after filling of the colon, when the films are viewed wet, if it is felt that there is a suspicious area, the patient still can be brought back and the

double contrast study done. As with any method of double contrast study, there is no substitute for adequate preparation of the patient. Also the degree of success is largely dependent upon how evenly the mixture has been distributed. With experience in rotating the patient and thus adequately distributing the mixture, the quality of the examination improves. One very definite advantage is that, with the fear of precipitation gone, the element of haste is removed and more time can be taken for adequate distribution of the mixture.

SUMMARY

A method utilizing methylcellulose mixed with the barium enema solution to prevent precipitation of barium is presented. This makes it possible in the majority of cases to do both a barium enema and double contrast study of the colon on the same day.

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THE POSTERIOR OBLIQUE VIEW IN DISLOCATION OF THE HIP

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DISLOCATION or subluxation of the hip joint may be overlooked in the conventional roentgenograms of this articulation, made after acute injury. The head of the femur may be almost exactly superimposed upon the acetabulum, though posterior to it, and if there is only slight overlapping of the rim of the acetabulum by the head of the femur, its significance may be overlooked. Occasionally such patients whose dislocation or subluxation was not recognized at the time of injury are seen several months later, and by this time irreparable damage has occurred. On the other hand, subluxation may sometimes be erroneously suspected in the routine anteroposterior view of the hip, when the joint capsule is relaxed due either to traction or to other causes.

In these painful injuries of the hip any roentgenographic position that accurately demonstrates subluxation or dislocation

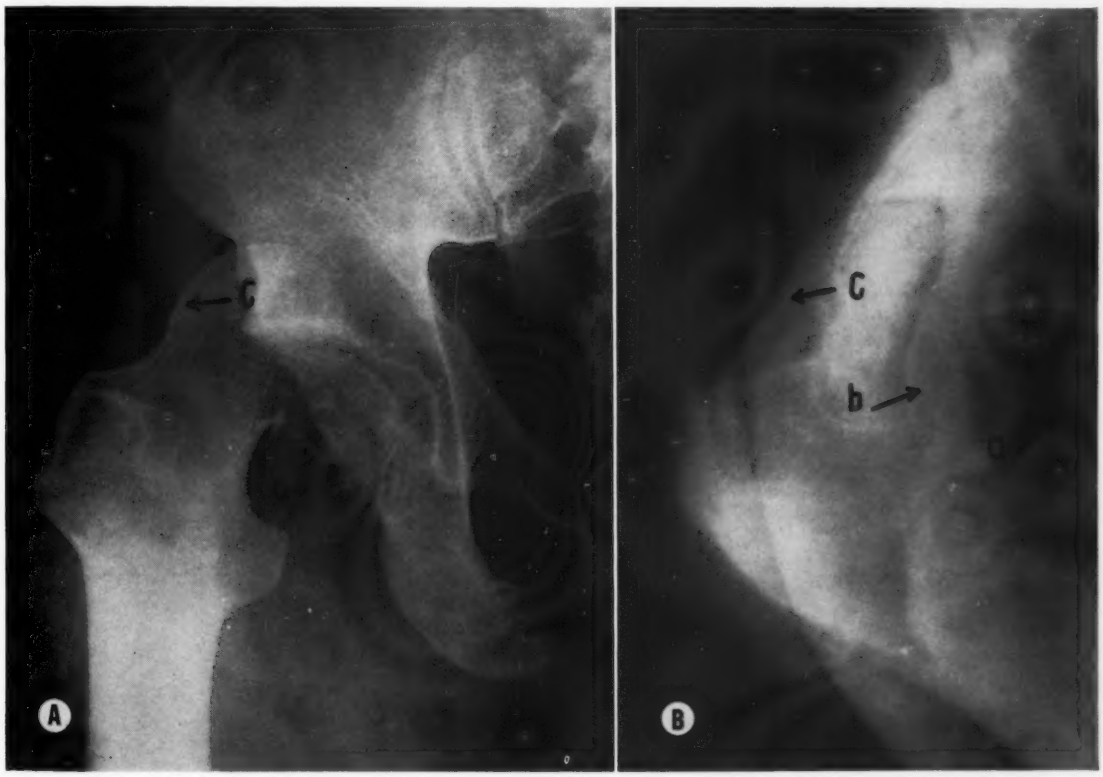

FIG. 1. (*A*) and (*B*) W.D.S., a forty-seven year old obese white male, was injured in an automobile accident on November 24, 1949. He sustained a posterior dislocation of the head of the left femur. A large fragment of bone was detached from the anteroinferior portion of the head of the femur and remained in the acetabulum. The dislocation was reduced by the closed method and the fragment of the head, about one-third of the size of the head, was subsequently removed from the acetabulum by open operation. (*a*) Detached fragment from head of femur; (*b*) intact posterior rim of acetabulum; (*c*) dislocated head of femur.
(Figure 1*A* is reproduced from an article by B. H. White and T. J. D. Scanlan entitled "Fracture Dislocation of the Femoral Head," published in the *Southern Medical Journal*, July, 1952.)

FIG. 2. (*A*) and (*B*) C.M.G., a forty-three year old obese white male, was injured in an automobile accident on April 5, 1952. His right hip was injured. He was sent home from a local hospital to recuperate in bed. On June 15, 1952 he fell off the edge of his bed, with aggravation of the right hip injury. Roentgenograms at this hospital showed fracture of the posterior rim of the acetabulum. There was a lunate compression or indentation fracture of the anteromedial portion of the head of the femur with mushrooming of the head. The head of the femur was subluxated posteriorly. The fracture of the head was apparently caused by the head impinging on the rim of the acetabulum. Attempted closed reduction was unsuccessful. At open operation, the head of the femur had to be trimmed down before it could fit into the acetabulum. (*a*) Fragment from posterior rim of acetabulum; (*b*) fracture of head of femur.

with minimal manipulation of the patient helps to assure proper diagnosis and treatment. The projection of Hsieh[2,3] provides the above information and also gives excellent detail of the posterior aspect of the hip and adjacent structures. This projection is an anterior oblique view, made with the patient in the prone position. The unaffected side is elevated 35 to 40 degrees and the weight of the body rests on the side of the injured hip. Unfortunately, most patients with hip injuries, especially if the head of the femur is out of place, cannot tolerate any such position because of excruciating pain.

However, a posterior oblique view made with the patient on his back with the injured side elevated 45 to 60 degrees is easy to execute, causes relatively little pain and demonstrates the relationship of the head of the femur to the acetabulum. Because the injured part is rolled up and not close to the table, some distortion of the osseous structures on the roentgenogram is inevitable. If the roentgen tube is elevated as much as possible, distortion will be minimized and adequate detail obtained.

In addition to demonstrating whether the head of the femur is out of the acetabulum, this view also shows if there is a fracture of the posterior rim of the acetabulum, and the size and position of the detached fragments. Accompanying fractures of the head of the femur may also be clearly demonstrated, since this oblique projection separates the dislocated head of the femur from the pelvis which lies anterior to it. There may be detachment of a small fragment of bone from the head of the femur, compression or lunate indentation of the head which distorts the shape of the head, so that reduction of the dislocation without open operation may be impossible.

As emphasized earlier, the use of an an-

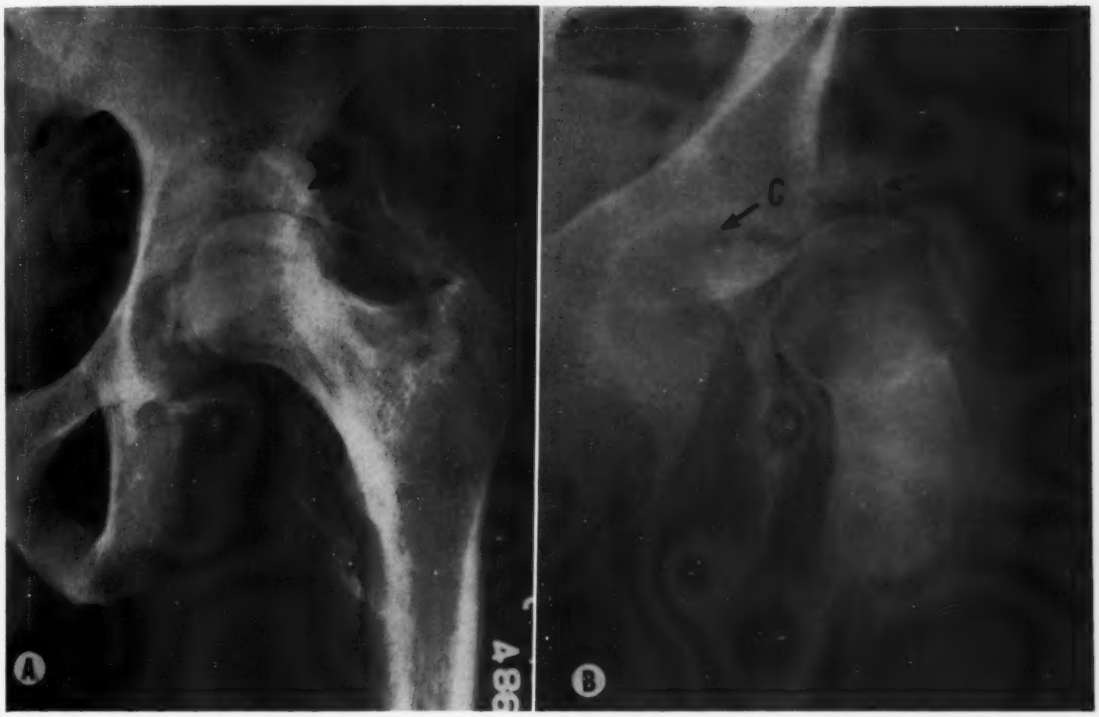

FIG. 3. (*A*) and (*B*) W.M.B., a twenty-six year old white male, was injured in an automobile accident on July 14, 1951. Roentgenograms made on November 23, 1951, showed an old dislocation of the right hip, fracture of the posterior rim of the acetabulum and traumatic myositis ossificans. (*a*) Fragment from posterior rim of acetabulum; (*b*) myositis ossificans; (*c*) superior margin of acetabulum.

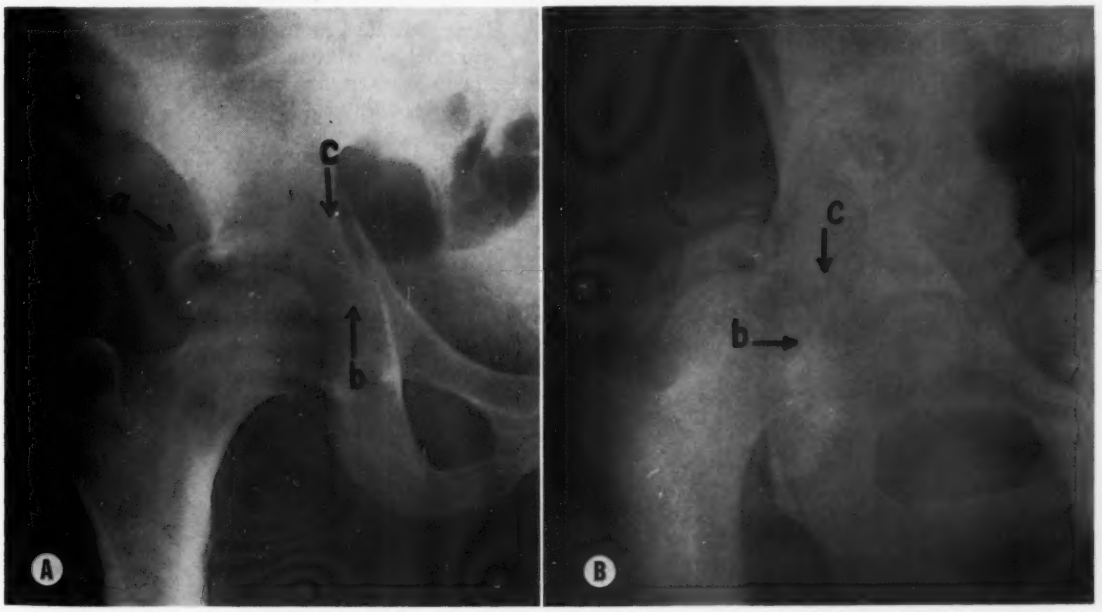

FIG. 4. (*A*) and (*B*) A.S.B., a thirty-two year old obese white male, was injured in an automobile accident on April 5, 1952. Roentgenograms showed a fracture of the acetabulum along the approximate line of the junction of the iliac bone with the ischial and pubic bones. There was also a comminuted fracture of the posterior rim of the acetabulum with posterior subluxation of the head of the femur, not obvious in the anteroposterior view. The subluxation was reduced and the fractures treated conservatively. (*a*) Fragments of posterior rim of acetabulum; (*b*) subluxation of head of femur; (*c*) fracture of acetabulum near junction of iliac bone with ischial and pubic bones.

terior oblique projection with the patient on his abdomen and his weight resting on the injured hip actually provides superb roentgenographic detail, but is impracticable in the acutely injured patient. A posterior oblique view similar to the one advocated here is mentioned by Clark[1] as "an unusual position applied in special circumstances when additional information is required regarding the posterior aspect of the iliac bone." This view has also been described by Urist[4] as giving a true picture of the displacement of the rim fragments since the posterior portion of the acetabulum is placed in profile. It is desired to stress its value as a relatively painless method of ascertaining definitely dislocation or subluxation of the hip, fractures of the posterior rim of the acetabulum and head of the femur in the acutely injured patient. It is not implied that this position replaces the conventional anteroposterior view, but rather supplements it.

SUMMARY

1. In acute injuries of the hip, a posterior oblique position with the patient supine and the injured side elevated 45 to 60 degrees, conclusively demonstrates dislocation or subluxation and associated injuries such as fracture of the posterior rim of the acetabulum or fracture of the head of the femur.

2. The injured patient can easily be placed in this position, in contradistinction to the anterior oblique view of Hsieh, which is very painful in dislocation of the hip but provides somewhat better roentgenographic detail.*

Veterans Administration Hospital
Montgomery 10, Alabama

* The illustrations were prepared by John L. Templeton, Medical Illustration Laboratory, Veterans Administration Hospital, Montgomery, Alabama.

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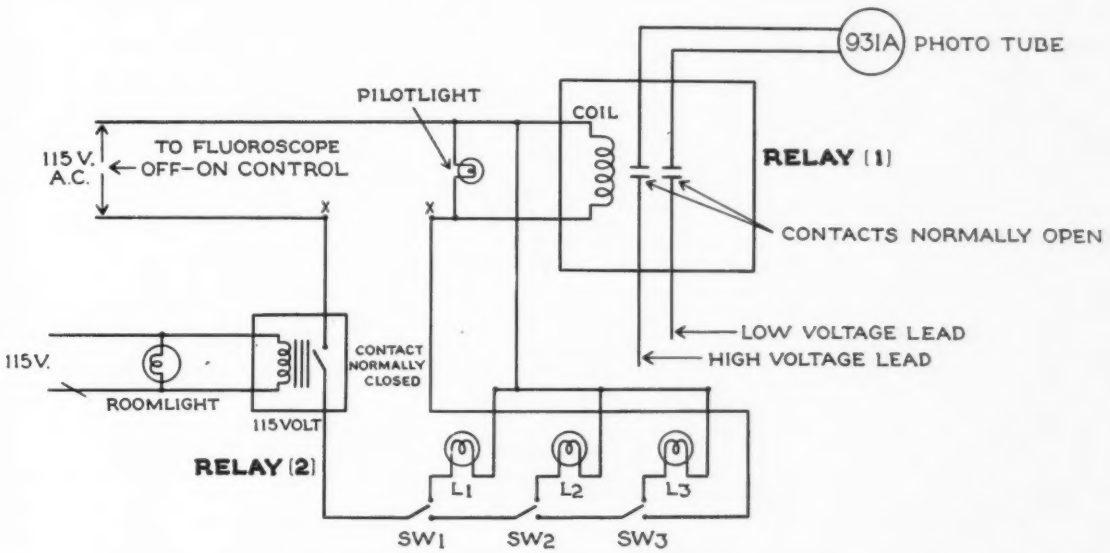
Vol. 72, No. 6

A WAY TO PROTECT AND LENGTHEN THE LIFE OF PHOTOTUBES IN FLUOROSCOPIC PHOTOTIMERS*

By LEE B. LUSTED, M.D.
SAN FRANCISCO, CALIFORNIA

WHEN daylight or artificial electric light enters the fluoroscopic room while the control switch for the fluoroscope is turned to the "ON" position, the phototube may be badly damaged or destroyed. This article describes a simple interlock system that will prevent this accident.

Many fluoroscopic spot film devices use a 931A phototube in the phototimer. This phototube contains a photosensitive cathode which may be damaged by light falling upon it while the high voltage is applied. The phototubes have a decreased life span when they are so exposed. The cost of a 931A replacement varies from $15 to $80 depending on the type of fluoroscope. Therefore, the circuit should be so arranged that turning on the electric lights or opening any door or window shutter will automatically cut out the high voltage to the phototube. Two fluoroscope models are known to have a protective device incorporated in the phototimer circuit. The protection system proposed uses a relay in the electric light circuit and microswitches which are mounted at all doors and windows in the fluoroscopic room. The microswitches are wired in series and so connected to the phototube circuit that when any window, shutter, or door is opened the voltage to the phototube is cut off automatically. The relay, in parallel with the room light, likewise opens the phototube circuit when the room light is on.



SW1---SW3 ARE MICRO SWITCHES ON DOORS, WINDOWS, ETC.
L1---L3 ARE INDICATOR LIGHTS TO SHOW WHICH SWITCHS ARE OPEN.
PILOTLIGHT IS ON WHEN PHOTO TUBE CONTROL CIRCUIT COMPLETED.

FIG. 1

Ordinarily, voltage is applied to the 931A phototube when the main fluoroscopic

* From the Department of Radiology, University of California School of Medicine, San Francisco, California.

switch is turned to the "ON" position. The fluoroscopic control does not need to be in the "X-Ray" position to have the tube active.

The accompanying simplified schematic diagram (Fig. 1) shows one method of connecting the microswitches. The installation can be made readily by the service representative.

Both the high and low voltage supplies to the 931A must be disconnected to protect the tube. One known phototimer model has a single relay whose contacts control both these voltages. The series of microswitches SW_1-SW_4 are inserted in the relay control circuit at X-X. When any one of the microswitches is activated by an opened door or window shutter, the relay is deactivated and the power to the 931A is cut off.

If there is no single relay (relay 1 in accompanying diagram) as mentioned above in a particular phototimer, the microswitches may be inserted in the circuit which does control the 931A voltage.

Department of Radiology
University of California Hospital
San Francisco 22, California

Vol. 72, No. 6

THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

Editor: Lawrence Reynolds, M.D. *Assistant-to-the-Editor:* Ruth Bigelow, B.S.

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Representatives on National Committee on Radiation Protection: Edith H. Quimby, New York, N. Y., Theodore P. Eberhard, Philadelphia, Pa.

Thirty-seventh Annual Meeting: Shoreham Hotel, Washington, D. C., April 21–23, 1955.

EDITORIAL

RADIOLOGY AND THE NATIONAL RESEARCH COUNCIL*

By EUGENE P. PENDERGRASS, M.D.

THE charter of the National Academy of Sciences, passed by Congress and approved by President Lincoln in 1863, provides that,[1]

"... the Academy shall, whenever called upon by any department of the Government, investigate, examine, experiment, and report upon any subject of science or art, the actual expense of such investigations, examinations, experiments, and reports to be paid from appropriations which may be made for the purpose, but the Academy shall receive no compensation whatever for any services to the Government of the United States."

Under this provision the Academy has acted since the time of its establishment as an official adviser of the Government on a wide variety of questions.[1]

With the development of World War I, military and naval problems needed solution, so the Academy, at the request of President Wilson, established the National Research Council as the active agent of the Academy to assist the Government in organizing the scientific resources of the country.[1]

As the work of the National Research Council progressed, it became evident that the Council might be a useful organization for peace time, and it seemed desirable that a more formal statement of the objects of the Council be made by the President together with an expression of his desire that the Council be perpetuated by the National Academy and assured of the permanent cooperation of the various departments of the Government. The President's recognition of this led him to issue, on May 11, 1918, the following Executive Order:[1]

"EXECUTIVE ORDER"

"The National Research Council was organized in 1916 at the request of the President by the National Academy of Sciences, under its congressional charter, as a measure of national preparedness. The work accomplished by the Council in organizing research and in securing cooperation of military and civilian agencies in the solution of military problems demonstrates its capacity for larger service. The National Academy of Sciences is therefore requested to perpetuate the National Research Council, the duties of which shall be as follows:

"1. In general, to stimulate research in the mathematical, physical and biological sciences, and in the application of these sciences to engineering, agriculture, medicine and other useful arts, with the object of increasing knowledge, of strengthening the national defense, and of contributing in other ways to the public welfare.

"2. To survey the larger possibilities of science, to formulate comprehensive projects of research, and to develop effective means of utilizing the scientific and technical resources of the country for dealing with these projects.

"3. To promote cooperation in research, at home and abroad, in order to secure concentration of effort, minimize duplication, and stimulate progress; but in all cooperative undertakings to give encouragement to individual initiative, as fundamentally important to the advancement of science.

"4. To serve as a means of bringing American and foreign investigators into active cooperation with the scientific and technical services of the War and Navy Departments and

* From the Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania. Presented at the Fifty-fourth Annual Meeting, American Roentgen Ray Society, Cincinnati, Ohio, Sept. 29–Oct. 2, 1953.

with those of the civil branches of the Government.

"5. To direct the attention of scientific and technical investigators to the present importance of military and industrial problems in connection with the war, and to aid in the solution of these problems by organizing specific researches.

"6. To gather and collate scientific and technical information, at home and abroad, in cooperation with government and other agencies, and to render such information available to duly accredited persons.

"Effective prosecution of the Council's work requires the cordial collaboration of the scientific and technical branches of the Government, both military and civil. To this end representatives of the Government, upon the nomination of the National Academy of Sciences, will be designated by the President as members of the Council, as heretofore, and the heads of the departments immediately concerned will continue to cooperate in every way that may be required."

(Signed) Woodrow Wilson
(No. 2859)

The White House
11 May, 1918

The administration of the Research Council is carried on by a small group of officers and an Executive Board. The Council itself is composed of eight major divisions, of which one concerns itself with international relations and the others are divisions of science and technology, representing respectively, physics, mathematics and astronomy; engineering and industrial research; chemistry and chemical technology; geology and geography; the medical sciences; biology and agriculture; and anthropology and psychology. With these divisions are associated various technical committees, appointed to take charge of projects undertaken by the Council.[1]

Financial support of the administrative work of the Council is derived from a gift of five million dollars to the National Academy of Sciences from the Carnegie Corporation of New York and from other special funds. For the support of the scientific projects undertaken or sponsored by it the Council relies upon special gifts and appropriations obtained from time to time from various sources.[1]

The Council is not an institution for the maintenance of scientific laboratories. It is rather an organization which, while clearly recognizing the indispensable value of individual investigation, hopes particularly to integrate the work of individual scientists and to assist in coordinating, in some measure, scientific attack in America upon large problems in the fields of scientific inquiry.[1]

The American Roentgen Ray Society is one of the affiliated societies of the Division of Medical Sciences of the National Research Council. In that an effort is being made to revitalize the relationships between the Division of Medical Sciences of the Council and its affiliated societies, it seems appropriate at this time to review some of the history of our affiliation as well as to give a short report on those activities of the Division which are of particular interest to radiologists.

At the annual meeting of the National Research Council held in Washington, D. C., May 3, 1947, the representative of the American Roentgen Ray Society, Dr. Barton R. Young, was impressed with the fact that there were no committees on radiology in the Council. Apparently, up to that time, there were no radiologic problems, or if so, they were handled by a subcommittee under the Committee on Surgery or some other major committee.

Dr. Young wrote the late Dr. Lewis H. Weed, Chairman of the Division of Medical Sciences, and learned that in order to create or form a committee on radiology, it was necessary for the Council to receive such a request from a federal agency. The radiologists of America should know that it was the representative of the American Roentgen Ray Society, Dr. Barton R. Young, who initiated the movement of getting the Council interested in what radiologists might contribute to science and to general welfare in medicine.

Shortly thereafter, Dr. Aubrey O. Hampton requested the Veterans Administration

to petition the Council for a Committee on Radiology. A Committee on Radiology was formed and this Committee has become increasingly active since its inception.

The Committee on Radiology, one of the medical advisory committees of the Council's Division of Medical Sciences, reviews applications in its realm of interest submitted to the Division for consideration. One of its most active functions has been to consider applications for grants and fellowships supported by the James Picker Foundation.

The James Picker Foundation was established in 1947 to provide funds to foster research in and broaden the scope of radiology. To advance these objectives the Foundation has made available a limited number of Grants in Radiologic Research.

Applications are reviewed by the Committee on Radiology. The recommendations of the Committee are transmitted to the Board of Directors of the James Picker Foundation for final determination of awards. The National Research Council thereupon carries out the administration of the funds and exercises such supervision of their use as will further the aims of the Foundation.

The Foundation and the Council welcome the cooperation of universities and research institutions in making known these opportunities to members of their staffs. Grants are not restricted to institutions within the United States.

Any project offering promise of improvement in radiologic methods of diagnosis or treatment of disease is eligible for assistance. The Foundation has expressed particular interest in the support of research oriented toward diagnostic aspects of radiology. It has specifically requested that none of its funds be used to subsidize research looking toward improvement of conventional radiologic apparatus, since that would fall within the province of commercial equipment manufacturers. However, if the development of a new technique is inseparably dependent upon the creation of a new apparatus for its performance, such activity would be deemed appropriate for support.

A limited number of Fellowships in Radiological Research also has been made available by the Foundation. Preference will be given to awards for work oriented toward diagnostic aspects of radiology.

PANEL ON RADIOLOGY, COMMITTEE ON GROWTH, DIVISION OF MEDICAL SCIENCES

In June, 1945, the National Research Council entered into a contract with the American Cancer Society in which it was agreed that the Council would make recommendations to the Society with respect to those proposed cancer research projects which in the judgment of the Council should be undertaken and financed by the Society. Within the Division of Medical Sciences, there was established the Committee on Growth appointed to review applications and fellowships in the field of cancer research and to provide to the Cancer Society consultative services and such benevolent supervision as will most effectively aid the investigators.

In order that the work of the Committee might be most effective, a series of specialty panels have been set up to aid in the review of projects. The Panel on Radiobiology reviews those applications for grants to support projects in the field of radiobiology and makes recommendations to the Committee after due consideration of these applications.

The interest of the Committee on Radiology in the fundamentals of radiobiology is broad and deep. In recognition of this, it is proposed to absorb the Panel on Radiobiology of the Committee on Growth into the Committee on Radiology and to give the latter the responsibility of serving the Committee on Growth.[2]

SUBCOMMITTEE ON RADIOBIOLOGY, COMMITTEE ON NUCLEAR SCIENCE, DIVISION OF PHYSICAL SCIENCES

This subcommittee was appointed to promote the study of the more basic aspects

in the natural sciences relating to radiation.

In June, 1950, the subcommittee sponsored a Symposium on Radiobiology, the Basic Aspects of Radiation Effects on Living Systems. The papers presented at this time have been published in a single volume by Wiley and Sons. In the preface to the volume, Dr. J. J. Nickson stated, "the purpose of the meeting was to present in orderly fashion the state of knowledge of the field at that time, and further to indicate the hiatuses that exist in our knowledge, particularly in areas that seemed susceptible of investigative attack."

Small conferences have been convened with no formal program arranged but with informal discussion only taking place. These have proved most helpful in pointing the direction for further research and study of radiation. The next small conference is scheduled for May, 1954. There will be an invited attendance of twenty-five scientists. Five men have been selected, each to lead a discussion for one-half day. These sessions have been received most enthusiastically by the participating men.

As an outgrowth of interest generated by the work of the Subcommittee on Radiobiology of the Committee on Nuclear Science, a new society, the Radiation Research Society, has been formed. This organization, with an initial membership of over 250 scientists, held its first business meeting in New York City on April 15, 1952. Its objectives have been stated as follows: (1) to promote original research in the natural sciences relating to radiation; (2) to facilitate integration of different disciplines in the study of radiation effects; and (3) to promote the diffusion of knowledge in these fields.

From this short report, it can be said that the physicians and scientists representing radiology are playing an increasingly important role in the activities of the National Research Council. For those of us in radiology, it should be understood that the privilege of representation in the Division of Medical Sciences is not static and that representation within areas of science shall be determined from time to time on the basis of (a) contributions by the society to the progress of science and its application in a broad sense and (b) the different needs of the Division.[2]

REFERENCES

1. National Research Council, Organization and Members, 1949–1950, Washington, D. C., December, 1949, pp. 2–5.
2. Division of Medical Sciences Annual Meeting, May 23, 1953, p. vi.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Pa. Annual meeting: Palmer House, Chicago, Ill., Sept. 20–23, 1955.

AMERICAN RADIUM SOCIETY
Secretary, Dr. R. E. Fricke, Mayo Clinic, Rochester, Minn. Annual meeting: Shoreham Hotel, Washington, D. C., April 21–23, 1955.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 713 E. Genesee St., Syracuse 2, N. Y. Annual meeting: Biltmore Hotel, Los Angeles, Calif., Dec. 5–10, 1954.

AMERICAN COLLEGE OF RADIOLOGY
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. T. Leucutia, Harper Hospital, Detroit 1, Mich.

FIFTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Secretary-General, Dr. Eugene P. Pendergrass, 3400 Spruce St., Philadelphia 4, Pa. Meets at Shoreham Hotel, Washington, D. C., April 24–29, 1955.

ALABAMA RADIOLOGICAL SOCIETY
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

ARIZONA RADIOLOGICAL SOCIETY
Secretary, Dr. R. Lee Foster, 507 Professional Bldg., Phoenix, Ariz. Two regular meetings a year. The annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. A. Norton, 843 Donaghey Bldg., Little Rock, Ark. Meets every three months and also at time and place of State Medical Association.

ATLANTA RADIOLOGICAL SOCIETY
Secretary, Dr. Albert A. Rayle, Jr., 478 Peachtree St., N.E., Atlanta, Ga. Meets monthly, except during three summer months, on second Friday evening.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. J. J. Daversa, 345 75th St., Brooklyn, N. Y. Meets monthly fourth Tuesday, October through April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Clayton G. Weig, 135 Linwood Ave., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse N. Y. Meets January, May, November.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary. Dr. F. A. Riebel, 15 West Goodale St., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Columbus Athletic Club, Columbus, Ohio.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. E. Burns Lewis, 670 N. Michigan Ave., Chicago 11, Ill. Meets second Thursday of each month, October to April inclusive at the University Club.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. H. F. Inderlied, 11311 Shaker Blvd., Cleveland 4, Ohio. Meetings at 6:45 P.M. on fourth Monday of each month from October to April.

COLORADO RADIOLOGICAL SOCIETY
Secretary, Dr. S. A. Patterson, Larimer County Hospital, Fort Collins, Colo. Meets third Friday of each month at University of Colorado Medical Center or at Denver Athletic Club.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. B. B. Alicandri, 20 Maple St., Springfield, Mass. Meets second Friday October and April.

DALLAS-FORT WORTH RADIOLOGICAL CLUB
Secretary, Dr. O. H. Grunow, 650 Fifth Ave., Fort Worth, Texas. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. F. Lang, Harper Hospital, Detroit, Mich. Meets monthly first Thursday, Oct. through May, at David Whitney House, 4421 Woodward Ave., at 6:30 P.M.

EAST BAY ROENTGEN SOCIETY
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. J. T. Shelden, Box 1021, Lakeland, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. Herbert M. Olnick, 417 Persons Bldg., Macon, Ga. Joint meeting with Florida State Medical Association, November, 1954.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary, Dr. R. D. Shapiro, 541 Lincoln Rd., Miami Beach, Fla. Meets monthly at various South Florida Hospitals.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. Harry Fishbein, 324 Medical Arts Bldg., Houston 2, Texas. Meets fourth Monday each month at Hermann Hospital.

IDAHO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Alfred M. Stone, St. Lukes Hospital, Boise, Idaho. Meets annually at same time as Idaho State Medical Association.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Stephen L. Casper, 1101 Maine St., Quincy, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. John A. Robb, 238 Hume-Mansur Bldg., Indianapolis 4. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

IOWA RADIOLOGICAL SOCIETY
Secretary, Dr. James T. McMillan, 1104 Bankers Trust Bldg., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY
Secretary Dr. A. M. Cherner, Hays, Kansas. Meets in spring with State Medical Society, and in winter on call.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. David Shapiro, 323 East Chestnut St., Louisville 2, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Solomon Maranov, 1450 51st St., Brooklyn 19, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Oscar Harvey, 3741 Stocker St., Los Angeles 8, Calif. Meets second Wednesday each month at Los Angeles County Medical Association Building, Los Angeles.

* Secretaries of societies are requested to send timely information promptly to the Editor.

Maine Radiological Society
Secretary, Dr. W. A. Russell, Augusta General Hospital, Augusta, Maine. Meets in June, October, December and April.

Maryland Radiological Society
Secretary-Treasurer, Dr. Paul W. Roman, 1810 Eutaw Place, Baltimore 17, Maryland.

Miami Valley Radiological Society
Secretary, Dr. G. A. Nicoll, 134 Apple St., Dayton, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. J. W. Walton, Horton Memorial Hospital, Middletown, N. Y. Meets 8:30 P.M., fourth Tuesday each month, September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. R. W. Byrne, 3321 N. Maryland Ave., Milwaukee 11, Wis. Meets monthly on fourth Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. John R. Hodgson, Mayo Clinic, Rochester, Minn. Meets three times annually, once at time of State Med. Assn., and in spring and fall.

Mississippi Radiological Society
Secretary, Dr. John W. Evans, 117 N. President St., Jackson, Miss. Meets third Thursday of each month at the Rotisserie Restaurant at 6:30 P.M.

Montana Radiological Society
Secretary, Dr. Grant P. Raitt, Suite 413 Medical Arts Bldg., Billings, Montana. Meets at least once a year.

Nassau Radiological Society
Secretary, Dr. Alan E. Baum, Hicksville, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska Radiological Society
Secretary, Dr. J. F. Kelly, Jr., 816 Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. Stanley M. Wyman, Massachusetts General Hospital, Boston, Mass. Meets third Friday of each month, October through May, at Hotel Commander, Cambridge, Mass.

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. Sidney Rubenfeld, 477 First Ave., New York 16, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Radiological Society
Secretary, Dr. W. C. Sternbergh, Charlotte Memorial Hospital, Charlotte, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. C. O. Heilman, 807 Broadway, Fargo. Meetings by announcement.

North Florida Radiological Society
Secretary, Dr. Ivan Isaacs, 1645 San Marco Blvd., Jacksonville, Florida. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. D. H. Baxter, Albany Hospital, Albany, N. Y. Meets in Albany area on second Wednesdays of October, November, March and April.

Northern California Radiological Society
Secretary, Dr. H. B. Stewart, 2920 Capitol Ave., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Ohio State Radiological Society
Secretary, Dr. M. M. Thompson, Jr., 319 Toledo Medical Bldg., Toledo 2, Ohio.

Oklahoma State Radiological Society
Secretary, Dr. John R. Danstrom, Medical Arts Bldg., Oklahoma City, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. F. C. Shipps, 214 Medical-Dental Bldg., Portland 5, Oregon. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. J. R. Raines, 214 Medical-Dental Bldg., Portland 5, Oregon. Meets annually in May.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. J. M. Converse 416 Pine St., Williamsport. Next annual meeting at Berkshire Hotel, Reading, Penna., May 20 and 21, 1955.

Philadelphia Roentgen Ray Society
Secretary, Dr. H. M. Stauffer, Temple University Hospital, Philadelphia 40, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Donald H. Rice, Western Pennsylvania Hospital, Pittsburgh 24, Pa. Meets second Wednesday of month, October through June at Hotel Roosevelt.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Paul W. Roman, 1810 Eutaw Place, Baltimore 17, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Section, Connecticut Medical Society
Secretary, Dr. William A. Goodrich, 85 Jefferson St., Hartford 6, Conn. Meetings are held bi-monthly.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Radiological Society of Greater Cincinnati
Secretary, Dr. Wm. R. Dickens, Cincinnati General Hospital, Cincinnati 29, Ohio. Meets monthly from September to June on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Dr. H. C. Chang, 1282 Emma St., Honolulu 13, T. H. Meets third Monday evening of each month at designated hospitals.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. J. T. Brierre, 700 Audubon Bldg., New Orleans. Meets annually during Louisiana State Medical Society Meeting.

Radiological Society of New Jersey
Secretary, Dr. Carye-Belle Henle, 195 North 7th St., Newark, N. J. Meets at Atlantic City at time of State Medical Society and in November in Newark, N. J.

Radiological Society of Southern California
Secretary, Dr. George Jacobson, 1200 N. State St., Los Angeles 33, Calif.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Henry H. Forsyth, Jr., 40 Meigs St., Rochester, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth St., Denver 7, Colo.

St. Louis Society of Radiologists
Secretary, Dr. W. B. Seaman, 510 South Kingshighway, St. Louis 10, Mo. Meets fourth Wednesday each month, except June, July, August and September.

SAN ANTONIO-MILITARY RADIOLOGICAL SOCIETY
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets Brook Army Medical Center Officers Mess first Monday of each month.

SAN DIEGO RADIOLOGICAL SOCIETY
Secretary, Dr. C. W. Bruner, 2456 Fourth Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary, Dr. I. J. Miller, 2680 Ocean Avenue, San Francisco 27, Calif. Meets quarterly.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. H. R. Morris, 1027 "D" St., San Bernardino, Calif.

SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Secretary, Dr. John A. Long, 1801 K St., N.W., Washington 6, D. C. Meets Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. George E. Irwin, Jr., 703 N. East St., Bloomington, Ill.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M. September to May inclusive.

SOUTH BAY RADIOLOGICAL SOCIETY
Secretary, Dr. H. R. Berman, 309 St. Claire Bldg., San Jose, Calif. Meets second Wednesday of each month.

SOUTH CAROLINA RADIOLOGICAL SOCIETY
Secretary, Dr. William A. Klauber, Self Memorial Hospital, Greenwood, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. G. K. Henshall, 311 Medical Arts Bldg., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas. Next meeting, Houston, Texas, January 21 and 22, 1955.

TRI-STATE RADIOLOGICAL SOCIETY
Secretary, Dr. S. N. Tager, 219 Walnut St., Evansville, Ind. Meets last Wednesday of Oct., Jan., March and May, 8:00 P.M. at Elks' Club in Evansville.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May, Service Memorial Institute.

UTAH STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

VIRGINIA RADIOLOGICAL SOCIETY
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

WASHINGTON STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Eva L. Gilbertson, 1317 Marion St., Seattle 4, Wash. Meets fourth Monday of each month from September through May at 610 Pine St., Seattle.

WEST VIRGINIA RADIOLOGICAL SOCIETY
Secretary, Dr. W. Paul Elkin, 515 Medical Arts Bldg., Charleston, W. Va. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

WESTCHESTER RADIOLOGICAL SOCIETY
Secretary, Dr. M. G. Priestman New Rochelle Hospital, New Rochelle, N.Y. Meets on third Tuesday of January and October and on two other dates.

WISCONSIN RADIOLOGICAL SOCIETY
Secretary, Dr. W. W. Moir, 309 Geele Ave., Sheboygan, Wis. Special meetings held during year; annual meeting each spring at Madison, Wis.

X-RAY STUDY CLUB OF SAN FRANCISCO
Secretary, Dr. Wm. W. Saunders, Veterans Administration Hospital, San Francisco 21, Calif. Meets monthly, third Thursday, at 7:45 P.M., Lane Hall, Stanford University Hospital.

CUBA, MEXICO, PUERTO RICO AND CENTRAL AMERICA

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA DE CUBA
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

SOCIEDAD MEXICANA DE RADIOLOGÍA, A. C.
Secretary General, Dr. Eugenio Toussaint, Calle del Oro No. 15, Mexico 7, D.F. Meets first Monday of each month.

ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA
Secretary, Dr. Rafael A. Blanes, Ave. Ponce de Leon 1175, Santurce, P. R.

SOCIEDAD RADIOLÓGICA PANAMEÑA
Secretary, Dr. L. Arrieta Sanchez, Apartado No. 86, Panama, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

BRITISH EMPIRE

ASSOCIATION OF RADIOLOGISTS OF THE PROVINCE OF QUEBEC
Secretary, Dr. J.-P. Jean, 4039 Tupper St., Westmount, Que.

BRITISH INSTITUTE OF RADIOLOGY INCORPORATED WITH THE RÖNTGEN SOCIETY
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 P.M.
Medical Members' meeting, on third Friday in each month at 5:00 P.M., 32 Welbeck St., London, W 1.

FACULTY OF RADIOLOGISTS
Honorary Secretary, Dr. J. F. Bromley, 45, Lincoln's Inn Fields, London, W.C.2, England.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

CANADIAN ASSOCIATION OF RADIOLOGISTS
Honorary Secretary-Treasurer, Dr. D. L. McRae, Suite 305, 1555 Summerhill Avenue, Montreal 25, Que. Meetings January and June.

SECTION OF RADIOLOGY, CANADIAN MEDICAL ASSOCIATION
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

SOCIÉTÉ CANADIENNE-FRANÇAISE D'ÉLECTRO-RADIOLOGIE MÉDICALES
General Secretary, Dr. Ls Ivan Vallée, 1058 rue St-Denis, Montreal 18, Canada. Meets third Saturday each month.

COLLEGE OF RADIOLOGISTS (AUSTRALIA AND NEW ZEALAND)
Honorary Secretary, Dr. Alan R. Colwell, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W.

SOUTH AMERICA

ASOCIACION ARGENTINA DE RADIOLOGIA
Secretary, Dr. Lidio G. Mocsa, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

SOCIEDAD ARGENTINA DE RADIOLOGIA, JUNTA CENTRAL, BUENOS AIRES
Secretary, Dr. Oscar F. Noguera, Santa Fe 1171, Buenos Aires. Meetings are held monthly.

SOCIEDAD ARGENTINA DE RADIOLOGIA, FILIAL DEL LITORAL, SEDE, ROSARIO
President, Dr. Francisco P. Cifarelli. Meets monthly second Wednesday, 19 ha 663 Italia St., Rosario, Santa Fe.

SOCIEDAD ARGENTINA DE RADIOLOGIA, FILIAL DEL SUR, SEDE, LA PLATA
President, Prof. Dr. Jose P. Uslenghi, La Plata. Meets monthly.

Sociedad Argentina de Radiologia, Filial del Centro Sede, Cordoba
President, Prof. Dr. David L. Caro, Corro 86, Cordoba. Meets monthly.

Sociedad Argentina de Radiologia, Filial del Norte, Sede, Tucuman
President, Dr. Julio Mendez Valladares, Rivadavia 358, Tucuman. Meets monthly.

Sociedad Argentina de Radiologia, Filial de Cuyo Sede, Mendoza.
President, Dr. J. A. Orfila, Hospital Central, Mendoza. Meets monthly.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Colombiana de Radiologia
Secretary, Dr. Luis E. Lozano B., Carrera 12 No. 20–69, 6º piso, Bogota, Colombia. Meets last Thursday of each month.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Sociedad de Radiologica, Cancerologia y Fisica Medica del Uruguay
Secretary, Dr. Arias Bellini, Av. Agraciada 1464, Piso 13, Montevideo, Uruguay.

Continental Europe

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Allies, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Société Française d'Electro-Radiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord
Secretary-General, Dr. A. Dariaux, 9 bis, Boulevard Rochechouart, Paris IXe, France.

Ceskoslovensk společnost pro rëntgenologii a Radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgen-Gesellschaft
Secretary, Dr. med. Peter Kröker, Evangelisches Krankenhaus, Huyssens Stiftung, (22a) Essen. Annual meeting, Sept. 5–8, 1954, Wiesbaden, Germany.

Societa Italiana di Radiologia Medica
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano. Torino, Italy. Meets biannually.

Nederlandse Vereniging voor Electrologie en Roentgenologie
Secretary, Dr. B. J. Rethmeier, Bornsestraat 378, Almelo, Netherlands.

Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10:30, X-Ray Dept.. Akademia Gdansk.

Wroclaw Section, Polish Society of Radiology
Secretary, Dr. R. Marciniak, Akad. Medyczna Wroclaw, 68 Curie-Skłodowska St., Wroclaw, Poland. Meets first Sunday of each month, X-Ray Department, Akademia Medyczna Wroclaw.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société Suisse de Radiologie)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

OAK RIDGE RESEARCH PARTICIPATION PROGRAM

University faculty members are invited to apply for places in the *Oak Ridge Research Participation Program* carried out by Oak Ridge National Laboratory and the Oak Ridge Institute of Nuclear Studies. Through this program, faculty members may conduct research in Oak Ridge for periods of three months to a year.

Oak Ridge National Laboratory offers opportunities for fundamental and applied research in physics, chemistry, metallurgy, biology, mathematics, and engineering, with a number of nuclear reactors and particle accelerators as the principal research instruments. The UT-AEC program is concerned with the internal and external effects of radiation on farm animals. The Institute Medical Division conducts extensive studies of the internal and external uses of radioisotopes in cancer treatment, and studies in radiation physics. In the Institute Special Training Division are opportunities for research in instrumentation, radioisotope applications, and the occurrence of radioactivity in ores and sea water.

Payment while a person is on leave for research participation approximates his university salary. Application should be made six months in advance of the time when the applicant would expect to begin his Oak Ridge research. Application forms and additional information may be obtained from the University Relations Division, Oak Ridge Institute of Nuclear Studies, P.O. Box 117, Oak Ridge, Tennessee.

MAINE RADIOLOGICAL SOCIETY

At a recent meeting of the Maine Radiological Society, the following were elected as officers for the year 1954–55: *President*, Dr. Clark F. Miller; *Vice President*, Dr. G. E. Clifford Logan; *Secretary-Treasurer*, Dr. Walter A. Russell, Augusta General Hospital, Augusta, Maine.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

At the annual meeting of the Rocky Mountain Radiological Society held in Denver, Colorado, on August 19, 20 and 21, 1954, the following officers were elected for 1954–1955: *President*, Dr. Alfred M. Popma, Boise, Idaho; *President-Elect*, Dr. Maurice D. Frazer, Lincoln, Nebraska; *1st Vice President*, Dr. Thomas J. Kennedy, Denver, Colorado; *2nd Vice President*, Dr. Charles F. Ingersoll, Denver, Colorado; *Secretary-Treasurer*, Dr. John H. Freed, 4200 East 9th Ave., Denver, Colorado.

AMERICAN BOARD OF RADIOLOGY

The deadline for filing applications for the Spring, 1955, examination by the American Board of Radiology is December 1, 1954. The examination will be held during the week of May 22, 1955, at the Palmer House in Chicago, Illinois.

Those candidates who will complete the required three years' training by June 30, 1955, will be eligible to appear for examination in May and those candidates who will complete their training by December 31, 1955 will be eligible to appear at the Fall examination which will be held the week of December 4, 1955 at the Palmer House in Chicago, Illinois.

B. R. KIRKLIN, M.D.
Secretary-Treasurer
429 First National Bank Building
Rochester, Minnesota

EASTERN CONFERENCE OF RADIOLOGISTS

The Eastern Conference of Radiologists will hold its Annual Meeting March 3, 4, 5, 1955, at the Sheraton-Plaza Hotel, Boston, Massachusetts. The New England Roentgen Ray Society, as host, warmly invites the attendance of all radiologists and their guests.

A program for Friday and Saturday morning, stressing newer concepts in diagnosis and therapy, is being arranged. A banquet will be held on Friday evening. For additional information, address Dr. Stanley M. Wyman, Secretary, New England Roentgen Ray Society, Massachusetts General Hospital, Boston, Massachusetts.

Vol. 72, No. 6

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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RADIOISOTOPES

Black, B. Marden, Woolner, Lewis B., and Blackburn, Charles M. The uptake of radioactive iodine by carcinoma of the thyroid gland: a study of 128 cases. *J. Clin. Endocrinol. & Metabol.*, Nov., 1953, *13*, 1378–1390.

From a review of the 128 cases of carcinoma of the thyroid seen at the Mayo Clinic in which radioiodine was employed diagnostically or therapeutically, a correlation between histologic type and iodine accumulation was obtained.

Twenty of the 23 lesions classified as the follicular type of papillary adenocarcinomas, or as either the follicular type or mixed follicular and solid type of malignant adenomas concentrated iodine, or could be stimulated to do so by ablation of the functioning thyroid tissue and administration of thiouracil.

Fourteen of 28 cases of mixed follicular and papillary type of adenocarcinoma accumulated iodine to variable degrees.

The papillary type of papillary adenocarcinomas, solid and Hürthle-cell types of malignant adenoma, and anaplastic adenocarcinomas almost never concentrated or could be stimulated to concentrate iodine.—*R. Kenneth Loeffler, M.D.*

Yuhl, Eric T., and Stirrett, Lloyd A. Clinical evaluation of the hepatic radioactivity survey. *Ann. Surg.*, Dec., 1953, *138*, 857–862.

This report gives the results obtained by the hepatic radioactivity survey in 283 patients. The details of the techniques are not given, but the authors refer to a previous report. The method employs intravenous injection of a tracer amount of I^{131} human serum albumin and the subsequent detection of areas of increased activity in the liver. Successive counts over coordinated areas of the thorax and abdomen are compared with normal values determined in persons without disease, and areas of increased radioactivity are considered to represent sites of neoplastic disease.

In 187 patients with proved primary neoplasms but no hepatic metastases, an over-all diagnostic accuracy of 96 per cent was obtained in that no foci of increased radioactivity were found. In 53 patients with known hepatic metastases demonstrated at laparotomy, the diagnostic accuracy in detecting an increase in the radioactivity over the metastases was 93 per cent. Conventional liver function tests in this latter group showed an accuracy of only 43 per cent.

Factors which influenced the interpretation of the survey were the presence of intraperitoneal fluid, inflammatory processes in the liver, and intra-abdominal inflammatory lesions such as active peptic ulcers. When such lesions are present, false positive results may be obtained. False negative results were obtained in patients who had small metastases, or metastases which lay deep within the liver.—*John P. Storaasli, M.D.*

Kaylor, C. T., and Van Cleave, C. D. Radiographic visualization of the deposition of radioberyllium in the rat. *Anat. Rec.*, Nov., 1953, *117*, 467–481.

Acute beryllium poisoning has been reported by previous workers to be accompanied by midzonal necrosis of hepatic cells, necrosis of the distal parts of the convoluted tubules of the kidneys, and hematopoietic damage with anemia and leukocytosis. To determine whether the damaged organs were the localization sites of beryllium, and also to determine the more detailed pattern of deposition within these organs, the authors employed a radioautographic technique.

Beryllium-7 was administered to 200 gram female rats as carrier-free, isotoic solutions of the sulfate chloride, or citrate at pH 4.5–5.0. Doses ranged from 1.5 to 11.2 millicuries, all except the largest given intravenously, usually in one injection. The largest dose was administered intraperitoneally over a one week period. Results are reported on animals sacrificed from two hours to sixty-two days after injection. To obtain adequate autographs from the gamma radiation of beryllium-7, a gold or platinum foil was interposed between tissue and emulsion.

Following administration of the chloride or sulfate, beryllium was found in the liver, spleen, skeleton and kidneys. The periphery of the liver lobule, the red pulp of the spleen, the cortex of the kidney, and the osteoid tissue adjacent to the epiphyseal plate in the distal end of the femur, were regions of greatest concentration.

Beryllium citrate behaved quite differently, with the skeleton being the only consistent site of deposition. The one reproduced radioautograph demonstrates activity in the spongiosa adjacent to the epiphyseal plate, with no detectable activity in the epiphyseal plate itself.

These data are generally consistent with those obtained from chemical assay.—*R. Kenneth Loeffler, M.D.*

Sherman, Alfred I., and Ter-Pogossian, Michel. Lymph-node concentration of radioactive colloidal gold following interstitial injection. *Cancer*, Nov., 1953, *6*, 1238–1240,

Colloidal sols, when injected interstitially, are collected by phagocytes and transported through the lymphatic pathways to the regional lymph nodes where they are trapped and concentrated. This is also one of the pathways for the spread of cancer and, since cancer can be destroyed by irradiation, it is logical to use a radioactive colloid in this manner to

irradiate tumor metastases selectively along the pathway of spread and in lymph nodes.

The colloid to be used should fulfill certain requirements. (1) The particles should be of a size and nature so as to induce phagocytic activity. (2) The sol should be chemically inert and physiologically nontoxic. (3) The radioactive element used should have a half-life sufficiently short to prevent irradiation over a prolonged period. (4) The amount of colloid concentrated in the lymph nodes must deliver a cancerocidal dose of radiation.

Radioactive colloidal Au^{198} fulfills requirements (1), (2) and (3). The metallic gold particles are of 50 to 250 μ in diameter suspended in a weak gelatin solution. The half-life is 2.7 days and the radiation consists of beta rays of 0.9 mev. and of gamma rays of 0.41 mev.

The gold is phagocytosed by the reticuloendothelial system and concentrated in the regional lymph nodes. It is well tolerated and no long term complications have been observed. However, because of the relatively short life of Au^{198} the rate of concentration in the lymph nodes is of great importance in determining whether or not an effective dose of radiation is delivered. Another factor of importance is the effect of massive infiltration of the lymph nodes and lymphatic channels by tumors which may block the drainage of gold in these areas.

Two groups of experiments on rabbits were done to determine the rate of concentration in regional lymph nodes of interstitially injected colloidal gold. Radioactivity determinations of regional lymph nodes at various times were made with a scintillation counter. Some rabbits were injected subcutaneously abdominally and others intraparametrially.

These experiments demonstrated effective and rapid drainage of the colloid to the regional lymph nodes with the maximum level of concentration within three to six hours for subcutaneous injection and six to ten hours for intraparametrial injection.

The total amount of activity concentration per unit weight of lymph node at a given distance from the site of injection is nearly constant and probably proportional to the total amount of gold injected. Calculations based on the concentration of activity in the lymph nodes refer only to the irradiation contributed by the beta rays which comprises the majority of irradiation from Au^{198}. The dose of radiation delivered per millicurie Au^{198} injected in the inguinal lymph nodes was 20,000 rep and in the iliac and preaortic lymph nodes 5,000 rep.—*Frank J. Rigos, M.D.*

Kanitz, Helmut Richard, Pfander, Friedrich, and Poppe, Hanno. Experimentelle Untersuchungen zur Frage einer lokalen Tumortherapie mit Radiogold. (Experimental investigations of local tumor therapy with radioactive gold.) *Strahlentherapie*, 1953, *91*, 208–222.

Radioactive gold chloride solution injected at the same time, just before, or just after a solution of the sodium salt of l-ascorbic acid into tissues of guinea pigs yields a precipitation of the radioactive metallic gold which remains relatively fixed in those tissues. A negligible quantity, approximately 1 per cent, of the gold injected is carried away from the injection site. In comparison with injected colloidal gold, only about one-tenth as much of the gold from the gold chloride is transported into the reticuloendothelial system.

In clinical use, local anesthesia is used to avoid the pain from the brief, but intense, local pH and sodium chloride concentration changes.—*Henry G. Moehring, M.D.*

Seaman, William B., Sherman, Alfred I., and Bonebrake, MacDonald. Radioactive gold in the treatment of malignant effusions. *J.A.M.A.*, Oct. 17, 1953, *153*, 630–633.

This article is a report on the use of Au^{198} in the treatment of malignant disease complicated by pleural effusion and ascites.

Of 40 patients with pleural effusion, approximately 50 per cent obtained some palliation. The dose in most cases was 100 millicuries. First patients had bilateral intrapleural injections at one sitting. Retreatment was done in three months in 2 cases. Eight patients received radioactive gold in both the pleural and peritoneal cavities. Benefit at times was obtained in one cavity and not in the other. Lack of immediate response does not necessarily mean that the treatment has failed.

Forty-two patients with ascites were treated, with palliation in 20 achieved.

This mode of therapy has been well tolerated in most instances. Mild nausea, anorexia and low grade fever were noted in a few cases. Some danger exists, however, from irradiation of the hemopoietic tissue. This occurs from the small portion of Au^{198} that gets into the blood stream from which it is later removed by the liver, spleen and bone marrow. This, plus the irradiation of the marrow by the gamma emission from Au^{198}, may result in aplastic anemia. In the authors' series, nothing more serious than a mild leukopenia was noted.

Hospitalization is required of all patients who receive more than 30 mc. of Au^{198}. The dose actually delivered to the serous membranes depends on area covered, depth, dilution and amount of Au^{198} used. A dose of 100 mc. into the adult peritoneal surface of 30,000 $cm.^2$ results in an average surface dose of 3,000 rep, assuming even distribution.

The authors conclude that the response to treatment is uninfluenced by the site of the primary tumor or the location of the effusion.—*G. E. Ross, M.D.*

Emerick, R. W., Holly, L. E., Joistad, A. H., Jr., and Corrigan, K. E. Diagnostic use of radioisotopes in a general hospital. *J.A.M.A.*, Feb. 6, 1954, *154*, 493–495.

The authors describe the radioiostope program in a small general hospital.

For the successful operation of such a program there must be demand for the services offered, trained personnel to administer and operate the department, requisite physical equipment and space, and adequate protection standards.

The laboratory is set up as a low level radiation laboratory utilizing dilute solutions obtained from a larger hospital or from commercial sources. Only diagnostic procedures are employed utilizing I^{131} and P^{32} with the former used in over 98 per cent of the studies.

The conditions investigated included thyroid hyperfunction and hypofunction, thyroid carcinoma, Legg-Perthes' disease, the differentiation of hypothyroidism and mongolism in infants and the localization of tumors. In all, 412 tracer studies were carried out over a period of four and one-half years.—*Leslie K. Sycamore, M.D.*

Peirce, Carleton B. Therapeutic value of radioisotopes in a general hospital. *J.A.M.A.*, Feb. 6, 1954, *154*, 495–499.

In considering the therapeutic use of radioactive isotopes, their characteristics and usefulness must be assessed either as substitutes for less available or less safe instruments presently at hand or as a means to appreciably advance the treatment of disease. The value of any physical agent or drug depends in some degree on its qualities and properties but more importantly on the knowledge, skill and intelligence of those who apply it. Isotopes are more dangerous for both the giver and the recipient than those agents that have been used previously and therefore a grave responsibility must be acknowledged by the administrative boards and the attending staff which sets up a program for their therapeutic use. No hospital should consider the establishment of such a program unless its medical staff is fully prepared to sacrifice time and funds, to submerge personal ambition and desire, to work closely as a team in the best interest of the patient, and to accept the responsibility of research in this as yet inadequately charted field.

The types of disease and number of patients suitable for treatment are limited; and of the known and probably existing 1,052 isotopes, only a few so far have been shown to be produceable in practical quantities with present facilities, to possess suitable and useful physical characteristics, and to be pharmacologically safe and physiologically useful. Those available for external therapy are phosphorus 32 and strontium 90 among the beta emitters, and cobalt 60 among the gamma sources. For intracavitary administration, sodium 24, phosphorus 32, cobalt 60, bromine 82, and gold 198 have been found useful. Consideration of the parenteral administration of radioactive substances must include study not only of the full gamut of the alpha, beta or gamma emission and the half-life of each substance but also specifically the pharmacological safety and biological activity or physiological progress through the tissues. So far only iodine 131 and phosphorus 32 have proved acceptable. Because of the many problems involved in the use of radioisotopes therapeutically available, personnel must include experts in clinical physiology, pharmacology, metabolism, hematology, endocrinology, radiation therapy and radiobiology, together with radiation physicists and a corps of trained technicians.

In the field of external irradiation, phosphorus 32 is useful in the treatment of superficial carcinoma of the skin, some keloids, and localized keratoses. Strontium 90 is of value in the treatment of benign vascular lesions of the conjunctiva and cornea. Cobalt 60 is a relatively ideal agent for the treatment of deep seated malignant lesions. Its use should be restricted, however, to those therapeutic centers that have at least 300 new cases of major malignant neoplasms per year and that have full staffs and qualified specialists.

For intracavitary irradiation, cobalt can be shaped into special applicators to fit an individual situation and then activated in the atomic "pile." In this form it is useful in the treatment of carcinoma of the cervix and the maxillary antrum and the urinary bladder. Sodium 24 or bromine 82 have been used in the balloon technique for the treatment of carcinoma of the bladder. In the serous cavities colloidal gold 198 or colloidal chromic phosphate incorporating phosphorus 32 have been utilized in achieving reduction of serous effusion in metastatic carcinoma. For interstitial irradiation, cobalt 60 and tantalum 182 are used as solid implants and colloidal gold 198 for direct injection. Intracavitary or interstitial treatment should find little place in a general hospital admitting less than 500 new patients with malignant disease a year.

Selection of an isotope for parenteral administration will depend on its degree of selective chemical affinity for the tissue to be influenced, the absence of material with a deleterious physical or chemical effect on other cells, tissue or the body as a whole, and the extent to which it will produce a beneficial result with less deleterious effect than some other therapeutic agent that may be available. In this field radioactive iodine meets virtually all the essential criteria in regard to thyroid disease. This isotope produces gradual depression of thyroid hyperfunction without damage to other tissues and with no surgical hazard nor toxic side-effect. It is particularly indicated in diffuse toxic goiter in patients over forty-seven years of age or in younger patients when antithyroid drugs fail and surgery is refused, in hyperthyroidism complicated by cardiovascular disease,

and in cases of recurrence after surgical intervention. Similar results can be produced with properly managed conventional roentgen therapy, and in the average general hospital there is not sufficient additional value in radioactive iodine therapy to warrant the required investment and additions to staff for that alone. I^{131} also has some value in the treatment of metastases from carcinoma of the thyroid. P^{32} is of value in the blood dyscrasias and malignant diseases: acute and chronic leukemia, polycythemia, the malignant lymphogranulomas and lymphosarcomas, and multiple myeloma. Here again, however, essentially the same results can be achieved by roentgen therapy.

It is concluded, therefore, that in only a few instances do radioactive isotopes afford a means whereby non-malignant and malignant disease may be controlled more easily or less expensively than with the older or more accessible methods. The entire study of radioactive isotopes as therapeutic agents is still in the realm of research and no general hospital should consider the institution of isotope therapy unless it treats 500 new cases of malignant disease or 100 of thyroid disease per year and is prepared to furnish the necessary personnel and physical facilities.—*Leslie K. Sycamore, M.D.*

Quimby, Edith H. The radioisotope program in the general hospital. *J.A.M.A.*, Feb. 6, 1954, *154*, 499–501.

The time is rapidly approaching when the radioisotope program in the general hospital will be taken as much for granted as any other laboratory service. The possible applications of isotopes include thyroid function studies, blood volume determinations, radioactive phosphorus therapy of leukemia and polycythemia, radioactive gold therapy for ascites, and radioactive iodine therapy for exophthalmic goiter. Other possible uses include the localization of brain tumors and liver metastases, determination of extracellular fluid volume, isotope clearance measurements for various tissues, studies of peripheral vascular disease, and various types of therapy.

Because of the very real danger inherent in the injudicious use of isotopes, the Atomic Energy Comission has set up certain restrictions. Every institution wishing to use radioisotopes is required to form an "isotope committee" including a hematologist; an internist; a person experienced in assay of radioactive materials and in protection of personnel against ionizing radiations; and if therapy is to be done, a therapeutic radiologist and qualified physicist. The person who is to take the responsibility for patients must have some firsthand knowledge and experience in isotope uses. He must be a physician in good standing and have had previous clinical experience with radiation or radioactive materials or be directly collaborating with a person possessing such training and experience. Minimum experience includes three months' active cooperation in a group using isotopes and a month of actually handling the isotopes and participating in the management of cases. The function of the radioisotope committee is to review applications for the use of isotopes primarily from a point of view of radiological safety but also in any advisory capacity which may be indicated. The radiation safety officer has the responsibility for maintaining radiation safety throughout the institution through setting up proper handling procedures and instructing all involved personnel in their use. The isotope laboratory will also need at least one technician trained in radioisotope technique. Such a technician may be trained on the job by any member of the isotope committee who possesses the required background.

For the actual functioning of the isotope laboratory applications must be submitted to the Atomic Energy Commission for each isotope. The first application must include a list of members of the isotope committee with their qualifications and an inventory of equipment available. In the integration of the isotope laboratory into the hospital organization, tests may be carried out in the same manner as other clinical tests. The result may be reported without interpretation or an interpretation may be furnished by the director. Therapeutic program should be incorporated into activities of the regular radiation therapy division.

In general, it is advisable that a radioisotope laboratory be started on a relatively small scale with provision for expansion of service and personnel as opportunity and experience permit.—*Leslie K. Sycamore, M.D.*

Stirrett, Lloyd A., Yuhl, Eric T., and Libby, Raymond L. The hepatic radioactivity survey. *Radiology*, Dec., 1953, *61*, 930–934.

The authors have made a radioactive survey on a total of 210 patients using iodinated human serum albumin (I-HSA) as the tracer agent and an especially adapted scintillation counter having a $\frac{3}{4}$ inch aperture. The survey is routinely made twenty-four hours after the single sterile intravenous injection of 300 microcuries of the test medium. Rechecks are made every twenty-four hours as necessary for confirmation of abnormally active areas, but not after the seventy-two hour period.

In 119 of the 151 patients with proved neoplasms, the liver appeared normal at laparotomy. The survey was 96 per cent correct in this group. In 32 patients hepatic metastases were found at laparotomy and the survey was correct in 94 per cent.

In their discussion, the authors point out that the hepatic radioactive survey is not contraindicated by the presence of icterus or cirrhosis without ascites. If ascites is present, the survey may be misleading. In the group with proved hepatic malignant involvement, they obtained two false negative surveys (2

patients out of 32) and in the 119 patients without hepatic involvement there were 5 false positive surveys, an accuracy of 96 per cent. The overall diagnostic accuracy was 95 per cent. It is felt that this type of survey is the most reliable test available for diagnosing hepatic neoplastic involvement.—*Dean Nichols, M.D.*

Seaman, William B., Ter-Pogossian, Michel M., and Schwartz, Henry G. Localization of intracranial neoplasms with radioactive isotopes. *Radiology*, Jan., 1954, *62*, 30–36.

Various investigators have employed radioactive isotopes to detect and localize brain tumors. Diiodo^{131}-fluorescein, K^{42}, radioactive iodinated human serum and radioactive sodium iodide are the chief substances which have been used in these studies.

The authors present an analysis of their findings in 200 patients studied for a long enough period to permit adequate follow-up and evaluation. A scintillation counter was used for radiation detection. Diiodo^{131}-fluorescein was injected intravenously in 0.25 millicurie amounts. Thirty-two positions on the skull were routinely surveyed in each patient.

Correct localizations were obtained in 30 of 65 patients with histopathologically verified tumors, an accuracy of 46 per cent. The radioactive isotope diagnosis of a localized lesion or absence of a focal lesion was correct in 10 of the 20 non-neoplastic cases, an accuracy of 50 per cent. In the entire group of verified cases, a correct localization of a focal lesion or the correct demonstration of the absence of a focal lesion was made in 40 cases (47 per cent). Subtentorial lesions were found to be much more difficult to localize than the supratentorial lesions. In 50 patients both radioactive isotope and electro-encephalographic studies were done. Results indicated that the two methods are somewhat complementary so far as localization of brain tumors is concerned.

Failure to localize a tumor may be the result of an unfavorable location or biologic characteristics that interfere with the basic mechanism of localization of the medium. The clinical status of this test is yet to be definitely determined, but the authors feel that it is a useful screening procedure to aid in the selection of patients for further diagnostic studies.—*Arno W. Sommer, M.D.*

Callender, Sheila T., Turnbull, Adam, and Wakisaka, Gyoichi. Estimation of intrinsic factor of Castle by use of radioactive Vitamin B12. *Brit. M. J.*, Jan. 2, 1954, *1*, 10–13.

In this study the authors checked the absorption of Vitamin B_{12} by labelling the substance with cobalt 60 and measuring the radioactivity in the feces after an oral dose of 0.5 microgram. In 10 normal patients 31 per cent of the radioactivity in the test dose was recovered from feces. In 13 patients with pernicious anemia 88.7 per cent was recovered. It was found that, by giving these pernicious anemia patients sources of intrinsic factor, the amount of test dose recovered of the labelled B_{12} was greatly reduced.—*T. D. Allison, M.D.*

Spangenberg, Harry D., Jr., and Pool, M. L. Production of roentgenograms by x-ray radiations obtained from radioactive x-ray emitters. *J. Am. Dent. A.*, Dec., 1953, *47*, 624–630.

The authors describe the production of roentgen rays by radioisotopes, either by the process of K-capture or by internal conversion of nuclear gamma rays. Using either method they envisage the use of these sources of roentgen radiation as a substitute for high voltage equipment in the production of roentgenograms.

The authors point out that such sources of radiation are minute and thus are suitable for placing within the mouth or other body cavity in order to visualize structures from positions heretofore impossible.

They reproduce two roentgenograms of the teeth and the jaw which were exposed by radiation from an isotope placed within the oral cavity of a skull. The isotope used was cesium, obtained by neutron bombardment from barium. Because the radioactivity was weak, the half-life being 9.6 days, the exposure times used were eleven hours and one week, respectively. They state that there are other isotopes which would have much greater activity and thus might be usuable in clinical practice for obtaining roentgenograms of the teeth or other structures.—*Arthur S. Tucker, M.D.*

Jarbak, J. R., Kamins, Maclyn M., and Vehe, Karl L. Radioautographic study of the deposition of calcium in the cranium, teeth and mandible. *J. Am. Dent. A.*, Dec., 1953, *47*, 639–648.

The incorporation of calcium in the skull, mandible, and teeth of a growing rat of unspecified age was studied by means of radioautographs. Five microcuries of Ca^{45} in 12.5 gm. of calcium as calcium chloride at pH 6.9 were administered intraperitoneally per 100 gm. of body weight. The animal was sacrificed one week after injection and prepared for radioautography.

Maximum deposition was in the sutures between the bones of the skull, in the mandibular condyle, in the walls of the pulp chambers of the incisor teeth and in the cementum of the molars. It is concluded that these are areas where calcium is being actively incorporated in the formation of new bone and teeth. Lesser concentration of radiocalcium was noted in the subperiosteum on all inner and outer tables of intramembranous bones, which is assumed to be due to ionic exchange. The presence of radiocalcium in a thin layer of coronal dentin suggests that ion ex-

change also takes place with saliva. Least uptake of radiocalcium was noted in the enamel of the molar teeth.—*R. Kenneth Loeffler, M.D.*

DRUMMY, W. W., JR. The use of radioactive iodine in the detection of thyroid dysfunction. *New England J. Med.*, Dec. 19, 1953, *249*, 970–973.

The author presents an analysis of 593 patients from a group of 2,000 in whom radioiodine was used for the evaluation of thyroid function. He uses the smaller group for study since the state of the thyroid function was established by criteria other than radioiodine uptake in this group.

The method of study consisted in the measurement of the accumulation of radioiodine in the thyroid region in a twenty-four hour period after the oral administration of a known amount of radioiodine.

A review of the 593 cases revealed 221 euthyroid patients, excluding those with goiter and those who had received iodine, thyroid medication, or antithyroid medication. The uptake in these patients ranged from 11 to 59 per cent of the administered dose (dosage figures not stated).

In 30 cases of myxedema there was no uptake greater than 15 per cent, and usually less than 5 per cent. Of the 70 cases of thyrotoxicosis 93 per cent had uptakes greater than 50 per cent.

Eleven of 41 patients with diffuse goiters accumulated more than 50 per cent of the radioiodine, whereas only 1 of 16 with nodular goiter exhibited any great avidity for iodine. There appeared to be some relationship between the size of the goiter and the radioiodine uptake.

High uptake values were noted in 21 patients who had been treated with antithyroid drugs and this affinity remained for one to six weeks after cessation of treatment.

Low values were observed after medication with thyroid or iodine, or iodine-containing radiopaque media. Low or normal values were found in patients with pituitary insufficiency.—*Denis V. Campbell, M.D.*

WOLLMAN, S. H., SCOW, R. O., and MORRIS, H. P. Radioiodide-concentrating ability of transplantable tumors of the thyroid gland in C3H mice. *J. Nat. Cancer Inst.*, Dec., 1953, *14*, 593–605.

Thyroid tissue accumulates I^{131} after injection into animals. Almost all of the I^{131} is bound in organic combination. The remainder, although only a small fraction of the thyroid I^{131}, is maintained at a concentration many times the radioiodide concentration of the serum. The ratio of radioiodide concentration in tissue to that in serum (T/S ratio) is a measure of the radioiodide-concentrating ability.

In the authors' series the T/S ratio was measured in several lines of transplanted tumors of the thyroid gland. It was determined when organic binding of radioiodide was permitted and when it was blocked by injection of propylthiouracil about one hour prior to radioiodide injection. The T/S ratio was much higher in dependent tumors (growth of tumor required host to be fed propylthiouracil), average 33, than in independent tumors, average less than 2, but never was as high as in the thyroid gland, ranging between 90 and 250.

Thus, the authors conclude that when thyroid tumor tissue becomes independent it appears to lose its ability to perform biochemical functions characteristic of thyroid tissue.—*Geno Di Bagno, M.D.*

DAILEY, MORRIS E., LINDSAY, STUART, and MILLER, EARL R. Histologic lesions in the thyroid glands of patients receiving radioiodine for hyperthyroidism. *J. Clin. Endocrinol. & Metabol.*, Dec., 1953, *13*, 1513–1529.

Histological examinations were performed on the thyroid glands of 23 patients who had received radioiodine by oral administration. The doses ranged from 0.4 to 21.8 millicuries, given from 12 to 558 days prior to obtaining the specimen. Two were postmortem specimens; the remainder were removed because of palpable nodules or because the clinical improvement was not sufficiently rapid following radioiodine therapy. Treatment in all cases was for hyperthyroidism.

Nine glands had lesions histopathologically compatible with Hashimoto thyroiditis. This incidence is greatly in excess of that to be expected in unirradiated glands. These lesions were found in glands that had received from 0.5 to 8.2 mc. 100 days or less before removal.

Those glands that had received larger doses at greater time intervals before examination showed marked atrophy and fibrosis, with nodularity in some glands which appeared to be due to attempted compensatory regeneration. It is felt that these changes are definitely the late effects of irradiation. Several of the remaining glands exhibited varying degrees of edema, fibrosis, diffuse follicular atrophy, and vascular change, which are considered to be radiation effects. It is noted, however, that equally pronounced deviations from normal are frequently observed in unirradiated hyperplastic glands.—*R. Kenneth Loeffler, M.D.*

BAUER, FRANZ K., GOODWIN, WILLIAM E., LIBBY, RAYMOND L., and CASSEN, BENEDICT. The diagnosis of morphologic abnormalities of the human thyroid gland by means of I^{131}. *Radiology*, Dec., 1953, *61*, 935–943.

A method of studying some of the morphological characteristics of the frontal profile of the human

thyroid gland in its normal and abnormal states is presented. The method uses I^{131} and the directional scintillation counter. The outline of the thyroid gland obtained is called a "scintigram."

A dose of carrier-free I^{131} ranging from 100 to 300 microcuries is administered orally in capsule form. The outlining procedure is done twenty-four to forty-eight hours later. The scanning tube is run over the neck or any area in which functioning thyroid tissue is thought to be present. The resulting tracing or "scintigram" is a record of the activity of the tissue examined.

Outlining the thyroid gland is used as a routine clinical procedure in the following instances: hyperthyroidism, simple goiter, solitary or multiple nodules, carcinoma of the thyroid gland and aberrant thyroid tissue.

Illustrative cases are presented.—*Arno W. Sommer, M.D.*

Krohmer, Jack S., Thomas, Charles I., Storaasli, John P., and Friedell, Hymer L. Detection of intraocular tumors with the use of radioactive phosphorus. *Radiology*, Dec., 1953, *61*, 916–921.

Studies were undertaken to determine the value of radioactive isotopes in the detection of intraocular tumors. P^{32} was chosen for the investigation because it is taken up in tumor tissue in considerably higher concentrations than in normal tissue. Five hundred microcuries of P^{32} in sterile isotonic saline were injected intravenously. Immediately after injection the counting procedure was started by placing the counting tube or crystal directly in contact with the eye, immediately adjacent to the suspected tumor. Additional counts were made at one-half and one hour after injection. Similar counts were made over an unaffected portion of the eye.

A total of 32 cases was studied and the selective uptake was recorded as the ratio of the count over the suspected site to that over a normal portion of the eye. It was concluded that a selective uptake ratio of 1.4 or higher in the anterior segment is very suggestive of tumors, while a selective uptake ratio of less than 1.2 over the anterior segment strongly indicates a non-neoplastic process. The uptake ratio for posterior tumors apparently was somewhat less reliable.

Included in the series are 9 proved anterior tumors, 3 proved posterior tumors and 14 non-neoplastic lesions. Six cases were not tabulated because a definite clinical or histopathologic diagnosis was not made.

The authors conclude that the P^{32} uptake studies are valuable adjuncts to careful clinical evaluation of suspected eye tumors rather than a conclusive diagnostic test.—*Arno W. Sommer, M.D.*

Stroebel, Charles F. Current status of radiophosphorus therapy. *Proc. Staff Meet., Mayo Clin.*, Jan. 13, 1954, *29*, 1–4.

The author analyzes the use of radiophosphorus in the treatment of chronic hemopoietic disease at the Mayo Clinic for the past twelve years.

It was found that certain diseases involving the bone marrow were affected only slightly, if at all, by radiophosphorus, these being mainly multiple myeloma, melanoma and many metastatic tumors to bone.

It was felt that roentgen rays are better for treatment of diseases, such as Hodgkin's disease, lymphosarcoma and chronic lymphatic leukemia, than radiophosphorus or nitrogen mustard.

The two main diseases that were most affected by radiophosphorus were chronic myelocytic leukemia and polycythemia vera. Twenty-two cases of chronic myelocytic leukemia were treated. Those treated with roentgen rays lived twenty-seven months, those by radiophosphorus thirty-three months after onset of symptoms.

Their primary use was in polycythemia vera. Three hundred cases were treated. A chart is given showing causes of death in 39. Hematological remissions lasting at least one year were obtained in 142 of 202 cases (70 per cent).

The dose for the simple polycythemia vera (without leukocytosis or myeloid immaturity) was an average of 5.7 mc. of radiophosphorus (range 3.0–21.0). Those with mature leukocytosis required an average dose of 8.3 and the leukemoid types 7.6 mc. (range 3.0 to 21.0).

Complications were hematological 12 per cent, vascular 4.6 per cent and miscellanous. Hematological complications were acute leukemia, hypoplastic anemia and anemia with leukemoid reactions.—*Leslie L. Lemak, M.D.*

Andrews, Gould A., Root, Samuel W., Kniseley, Ralph M., and Kerman, Herbert D. Intracavitary use of colloidal radioactive gold. *Radiology*, Dec., 1953, *61*, 922–929.

During the last two years the authors have been carrying on a program of experimental treatment with radioactive gold. They have injected the isotope into pleural and peritoneal spaces of patients with effusions caused by neoplasms and have studied its fate, distribution and effect. The results of treatment in 39 cases are summarized.

Before administering the isotope they aspirate a considerable portion of the fluid. The radioactive colloidal gold is introduced through a polyethylene tube, using a simple gravity infusion device similar to those used for giving intravenous fluids. Physiological saline is used for rinsing. During the first few hours after the gold has been given the patient must

move about frequently to aid free mixing in the cavity. The operator received radiation doses far below tolerance. Most of the patients treated recently were given 150 mc. for ascites and 75 mc. for pleural effusion. In a few cases the treatment was repeated several weeks after the initial dose. The optimal dosage of gold has yet to be determined.

Small but measurable amounts of radioactivity are found in the blood soon after the gold is injected. Urinary excretion is very slight. Less than 1 per cent of the total dose can be recovered from the urine. Autopsy studies of the liver, spleen and bone marrow after intracavitary administration indicate that a variable amount reaches these organs, and that the total may be as much as 15 per cent of the initial dose. Some of the treated patients experienced considerable nausea and vomiting from about the second to the fourth or fifth day after the treatment. In 16 of the 39 patients treated there was cessation of, or distinct decrease in, fluid accumulation. The most suitable cases for this therapy are those who have no large neoplastic masses, who are maintaining fair general health, and whose only symptoms are those related to the presence of fluid accumulations. Intracavitary gold treatment can be combined successfully with deep roentgen therapy of a local area of neoplasm, and it may be possible to give full therapeutic doses of both forms of radiation.—*Arno W. Sommer, M.D.*

CHEMOTHERAPY

Feinstein, Robert N., and Cotter, Gladys J. Lack of protective effect of allyl thiourea against x-irradiation. *Science*, Nov. 6, 1953, *118*, 552.

The authors found no biologic correlation between the ability of allyl thiourea to protect mice against the lethal effect of roentgen irradiation and its ability to protect polymethacrylic acid from radiation-induced loss of viscosity. Some of the drugs less effective in the polymer test system (sodium azide, cysteine) were more effective than allyl thiourea in protecting against radiation fatality. In some cases, the latter drug, though without toxicity itself, seemed to hasten the lethal result of roentgen irradiation.—*T. J. Wachowski, M.D.*

Shay, Harry; Zarafonetis, Chris; Smith, Nathan; Woldow, Irving; and Sun, David C. H. Treatment of leukemia with triethylene thiophosphoramide (thio-tepa); preliminary results in experimental and clinical leukemia. *A.M.A. Arch. Int. Med.*, Nov., 1953, *92*, 628–645.

Triethylene thiophosphoramide was used in treating 49 patients in a six month period. Thirty-nine cases had various malignant diseases of the hematopoietic system and 10 had other inoperable malignant neoplasms. Most of the cases of chronic myelogenous and lymphatic leukemia were found to respond favorably, whereas the acute leukemias did not. Several cases of Hodgkin's disease reported favorably. The cases of lymphoma did not respond as well as those of Hodgkin's disease. Two cases of recurrent and metastatic adenocarcinoma of the breast responded quite well as evidenced by disappearance of the metastatic axillary mass and gradual sloughing and healing of the recurrent malignant lesions. The healing process was complete in one case and partial in the other.

Triethylene thiophosphoramide is a suppressive drug, and supposedly the cytotoxic action is due to changes produced in the chromosomes especially of proliferating cells of all types, including nonmalignant cells. Bone marrow depression is the most significant toxic manifestation of this drug. If undue depression of bone marrow occurs, it can be reversed by withholding the drug. Triethylene thiophosphoramide, when given in the dosage as the authors give it, produces no nausea or vomiting, no pain when injected intramuscularly, and no venous thrombosis results when given intravenously. It is effective in some cases when given orally and in others it has to be administered either intramuscularly or intravenously. Patients with very high white blood cell counts should be kept on a high fluid intake and medication to keep the pH of the urine above 7, as rapid cytolysis may lead to formation of uric acid calculi.

The dosage schedule cannot be fixed due to variations in individual response. The best results are obtained by daily administration of the drug initially and this is guided by daily consideration of the hematological picture. Subsequently, the maintenance dosage can be established and the frequency of the blood counts can be reduced accordingly. There is a fairly wide range of safety between the effective therapeutic dose and the dose that unduly depresses the bone marrow.

Similar results were obtained in the treatment of chronic myelogenous (chloro) leukemia and acute monocytoid myelogenous leukemia in rats, with triethylene thiophosphoramide, as in humans. The drug was given by means of a stomach catheter and was found to be as effective as when given parenterally.

Representative cases are cited, including those showing good, limited, and no response to the drug. The technique and dosage for the cases are given.

The authors emphasize that this is a preliminary report with observations covering a six month use of triethylene thiophosphoramide. It is not known whether resistance will develop to the drug, as it is suppressive in action and not curative.—*Charles W. Cooley, M.D.*

Persky, Lester, and Austen, George, Jr. ACTH in radiation cystitis. *J. Urol.*, Nov., 1953, *70*, 724–728.

This is an evaluation of ACTH therapy for late radiation cystitis in 3 patients in each of whom there was bladder ulceration, fibrosis and symptoms of dysuria, nocturia and hematuria. Following treatment by intravenous or intramuscular administration of the drug, there was very little cystoscopic change noted. The symptoms of dysuria disappeared rapidly and the bladder capacity in all 3 individuals was doubled. Duration of the initial beneficial effect has varied from less than one week to several months, with gradual recurrence of symptoms of bladder irritability in 2 of the 3 patients.

This paper is presented with the hope that further investigation of ACTH may prove of some value in the earlier lesions of radiation cystitis and other forms of irradiation reaction.—*George W. Chamberlin, M.D.*

Storaasli, J. P., Rosenberg, S. A., and Friedell, H. L. The effect of cysteine on the radiosensitivity of rat lymphosarcoma. *Cancer*, Nov., 1953, *6*, 1244–1247.

Cysteine has been demonstrated to cause a large increase in the LD50 dose of total body roentgen treatment. It also decreases the lethal action of roentgen rays on malignant cells. With this in mind this study was undertaken to determine whether cysteine would decrease the radiosensitivity of normal tissues to a greater extent than that of malignant tumors and thus increase the therapeutic ratio.

Rats were implanted with a suspension containing Murphy-Sturm lymphosarcoma. One group of animals received cysteine in doses of 800 mg. per kg. of body weight given intraperitoneally ten to twenty minutes prior to irradiation.

Cysteine was found to decrease the radiosensitivity of this tumor. The tumors regressed in 25.6 per cent of the animals receiving cysteine prior to roentgen therapy, as compared to 70.3 per cent regression in the control group receiving only roentgen therapy.

A possible mode of action of cysteine in protecting against the lethal properties of radiation is that the high level of sulfhydryl compounds can protect vital sulfhydryl enzymes by competition for the oxidative radicals. A second possible mechanism of action is that there is a local tissue anoxemia produced by the metabolism of cysteine in vivo.

It would appear that the decreased radiosensitivity of the Murphy-Sturm lymphosarcoma in cysteine-treated rats is comparable to the decreased radiosensitivity of rats to total body roentgen irradiation.

It does not seem advisable to utilize cysteine as an adjunct to roentgen therapy in the treatment of patients with malignant neoplasms. It is logical to assume that normal tissue would be spared as well as tumor tissue so that an obvious alteration of the therapeutic ratio of the radiosensitivity of the tumor versus normal tissues would not occur. It is possible, however, that certain biological systems may be selectively protected with cysteine.

It is suggested that cysteine may protect the hemopoietic system when used in patients who are to receive nitrogen mustard therapy for lymphoma.—*Frank J. Rigos, M.D.*

Crichton, E. P., and Hamman, H. A. Typhoid osteomyelitis of spine treated with chloramphenicol. *Canad. M.A.J.*, Nov., 1953, *69*, 529–530.

This is a case report of a fifty-nine year old male who, following a prostatectomy, developed chills, fever and backache. The backache recurred subsequently and increased in severity. It was treated with cortisone. Later, roentgenograms showed destruction of the eighth dorsal vertebra with narrowing of the body and kyphosis. There was evidence of destruction of the adjacent vertebral surfaces and of an abscess extending from the sixth to the eleventh dorsal vertebrae.

The condition was tentatively diagnosed as Pott's disease and the patient was sent to a sanatorium. Here, the chills and fever gradually increased, and finally positive agglutination tests and blood culture for typhoid H were obtained. He was given a course of chloramphenicol and dramatic improvement followed. Roentgenograms five months later showed fusion of the adjacent affected vertebrae, and good new bone formation. The patient was able to resume normal activity.

The authors believe that the prostatectomy activated a pre-existing carrier state, and that the cortisone caused the exacerbation of the disease. This is one of the few cases of typhoid osteomyelitis treated with chloramphenicol and the first case in which drainage of the abscess was not necessary.—*George L. Sackett, Jr., M.D.*

Peabody, J. Winthrop, Jr., Buechner, Howard A., and Anderson, Augustus E. Hamman-Rich syndrome; analysis of current concepts and report of three precipitous deaths following cortisone and corticotropin (ACTH) withdrawal. *A.M.A. Arch. Int. Med.* Dec., 1953, *92*, 806–824.

Diffuse interstitial pulmonary fibrosis of the type originally described by Hamman and Rich is a disease of unknown etiology for which no specific therapy has yet been established.

Cortisone and corticotropin have been tried, with disappointing results. The authors employed the hormones in 3 patients, 1 manifesting the acute form of the disease, 1 a subacute form, and the third a chronic form. In all 3 patients, initial improvement was followed by a fulminant relapse and death.—*George Cooper, Jr., M.D.*

Scott, Roland B., and DeLilly, Mayo R. Idiopathic calcinosis universalis; report of a case in a child treated with corticotropin (ACTH) and cortisone. *A.M.A. Am. J. Dis. Child.*, Jan., 1954, *87*, 55–62.

Calcinosis is the term applied to the abnormal deposition of calcium salts into the skin, subcutaneous tissues, muscles and tendons. The disease is usually divided into two clinical types, depending upon whether the deposit is localized (calcinosis circumscripta) or generalized (calcinosis universalis). The latter type is the severer of the two and is the variety usually observed in children and young adults. The condition is uncommon, and only sporadic reports have appeared in the pediatric literature. The disease is characterized by remissions, exacerbations, and chronicity. Heretofore, treatment has been unpredictable and generally unsatisfactory.

Because of the reported beneficial results of cortisone therapy in other collagen diseases, this hormone was administered by the authors to their patient for a period of more than one year. Serial roentgenograms were made over a period of eighteen months. According to the roentgenograms, there was no significant improvement in the status of the soft tissue calcium deposits during the therapy.

Some of the serial roentgenograms are reproduced. —*Ralph S. Bromer, M.D.*

Waisman, Harry A., and Harvey, Roger A. Radiological evidence of growth in children with acute leukemia treated with folic acid antagonists. *Radiology*, Jan., 1954, *62*, 61–64.

Folic acid antagonists have been found to produce temporary bone marrow remissions in children with acute leukemia. Definite improvement in the osseous manifestations of the disease, and a definite increase in the average duration of life have been described.

The authors report the results of an evaluation of the roentgenographic findings in 43 patients who were treated with folic acid antagonists. A group of 21 patients with acute leukemia, treated before the advent of folic acid antagonist therapy, served as controls and showed roentgenographic changes in the osseous system characteristic of the disease. The antagonists-treated group exhibited a definite tendency to form transverse zones of diminished or increased density near the ends of the long bones. These roentgen changes were not considered specific but were regarded as evidence of temporary arrest of the disease as a result of the treatment. Their appearance generally coincided with clinical and hematological evidence of remission. Cases not treated with antagonists did not show these transverse bands.

The transverse bands are evidently a result of alterations in trabecular development due to competition for space with infiltrating and proliferating leukemia cells during the periods of active progression of the disease in the osseous system. Growth of bones apparently continued at a normal rate during prolonged periods of remission.—*Arno W. Sommer, M.D.*

MISCELLANEOUS

Lorenz, Egon, Congdon, Charles C., and Uphoff, Delta. Prevention of irradiation-induced lymphoid tumors in C57BL mice by spleen protection. *J. Nat. Cancer Inst.*, Oct., 1953, *14*, 291–301.

Previous work by others has shown that lead shielding of a part of the bone marrow or spleen in susceptible mice will significantly reduce the incidence of radiation-induced lymphoid tumors. It was concluded that these tumors were not the direct result of irradiation and it was postulated that prolonged depression of hematopoietic tissues may be an important factor in such tumor induction.

One hundred eighty-one two month old mice (C57BL) were evenly distributed into three groups. The first group had exteriorized spleens but no lead-shielding. The second group had lead shielding of the exteriorized spleens. These two groups were irradiated with four weekly doses of 225 r each (total body). The third group was irradiated with a single dose (900 r total body) with exteriorized, lead-shielded spleens. Tumor incidence of 68 per cent in the first group was compared to incidences of 2 per cent and 0 per cent in the remaining two groups, respectively.

It was concluded that protection of the spleen prevents induction of lymphoid tumors in these mice. Prolonged depresssion of bone marrow and lymphoid tissue apparently favors tumor development. The rationale of spleen shielding remains obscure.—*J. Maurice Swain, M.D.*

Chamberlain, Richard H. Radiation protection comes of age. *J.A.M.A.*, Oct. 3, 1953, *153*, 488–491.

Adequate knowledge regarding the hazards to human beings from ionizing radiation is now available and the nature of protective measures is reasonably well understood at the present time. Instruments and units for measurement of radiation are also available. The new era in radiation protection became possible by 1935 after the international acceptance of the roentgen as the unit of ionizing radiation.

A good deal of ungrounded fear has been generated as regards the potential dangers associated with the development of atomic energy and the increasing medical uses of radiation. The hazards from these sources have been far less than the benefits derived in the form of increased awareness of the problem. As a general rule, unnecessary radiation exposure with no hope of benefit should be discouraged. It is

well to recall that all humans are continually exposed to measurable levels of ionizing radiations in the form of cosmic radiation, and the emission of natural radioactive substances.

A radiation hazard is one of degree and quantity. The maximum permissible exposure to the whole body or gonads has been set at 0.3 r per week measured in free air (0.5 r on the body surface) or, in the case of the hands and forearms, at 1.5 r per week. These values refer to externally originating roentgen rays or gamma rays of energies less than 3.0 mev. These limits should never be exceeded unlesss such excess cannot reasonably be avoided in the case where the cause is for a meritorious purpose, such as in atomic warfare or in the military use of atomic energy.

The effectiveness of radiation protection in the United States is due to three factors: (1) information, basic and applied; (2) education and professional standards; and (3) methods of good practice.

Information has been accumulated and disseminated in recent years by the National Committee on Radiation Protection, articles in professional and technical publications, and the Atomic Energy Commission.

Education and professional standards offer the only valid method of ensuring good practice. In medicine, exceptional qualifications in these respects are attained by the radiologists in their training in radiation physics and radiobiology and by the group of radiological physicists which collaborates with them. Other physicians who need to use radiation in certain fields can and should receive training in the special problems of radiation protection. The Atomic Energy Act requires the Atomic Energy Commission to ensure that proper safety and health precautions are employed as a requisite to obtaining radioactive isotopes from the commission.

"The general plan in setting up a program for radiation protection will probably follow along these lines; (a) formation of a well-informed and well-trained group or acquisition of the same information and training by an individual; (b) establishment of a well-designed and properly protected installation with good equipment; (c) initial inspection and testing of the safety of the apparatus and radiation sources; (d) appraisal of the indications and levels for radiation esposure of all persons, including 'workers,' patient, and the general public; and (e) establishment of a regular system of monitoring with safety and health consultations with competent physicists."—*J. N. Ané, M.D.*

Braestrup, C. B., Green, D. T., and Snarr, J. L. Convergent beam irradiator; apparatus for convergent beam irradiation with stationary or moving source. *Radiology*, Oct., 1953, *61*, 614–624.

A convergent beam irradiator is described and illustrated. With this equipment a source of radiation may be freely moved over a large part of the surface of a sphere, with the beam always directed at the center of a sphere. Once a patient is fixed in position with the center of the tumor coinciding with the center of the sphere which defines the motion of the source, the beam is always pointed at the tumor, irrespective of the position of the source. A special treatment couch is an integral part of the equipment and is designed to facilitate set-up and to allow a wide choice of treatment methods.

The control equipment makes possible rotation of the source over a predetermined arc, with independent control of the arc over which the radiation is emitted.—*Arno W. Sommer, M.D.*

Sørensen, Hans Rahbek. A guide for use in x-raying the lumbar column in the lumbo-inguinal projection. *Acta orthop. Scandinav.*, 1953, *23*, 87–89.

The author describes in detail a plastic guide used in making roentgenograms of the lumbar spine in the lumbo-inguinal projection. This projection often provides information about lateral herniations of the nucleus pulposus. The guide was designed to simplify this procedure and improve the roentgenograms.

The author gives in detail the construction of the guide and directions as to its use.—*C. Peter Truog, M.D.*

Trautmann, Johannes, Frey, Julius G., and Schaaf, Johannes. Experimentelle Untersuchungen über die Wirkung kleinster Röntgendosen auf das Keimepithel des Rattenhodens; Beitrag zur Frage der Kombinationsbestrahlung nach Pape. (Experimental investigations of the effect of minimal roentgen radiation on the germinal epithelium of the rat's testes; a contribution to the problem of combined irradiation of Pape.) *Strahlentherapie*, 1953, *91*, 602–616.

The aim of these investigations was to test the validity of, and extend, the observations of Pape on the effect of small doses of roentgen radiation in the so-called "combined irradiation" (tiny preliminary doses followed by intensive irradiation). The measure of radiation effect was the histologic change in the germinal epithelium of irradiated rats' testes.

After studying a control group of non-irradiated rats' testes to establish an understanding of the normal variations in the germinal epithelium, these workers irradiated several series of rats with 300, 600, 1,000, 1,500 and 3,000 r (165 kv.; 0.35 mm. Cu filter; hvl. of 0.7 mm. Cu; 10×15 cm. field at 32.5 cm. f.s.d., 43 r/min.). The animals were sacrificed at the end of eight days except those which received 1,500 and

3,000 r; these rats died of radiation effects after 40–100 hours.

The final series of animals were subjected to teleroentgen dosage of 0.25 r daily for 80–100 days before they received massive doses of 600, 1,500 or 3,000 r. This irradiation with minimal doses constitutes the preliminary portion of the combined irradiation. These animals were also sacrificed at the end of 8 days, except that those given 3,000 r died earlier of radiation effect. However, all of the rats irradiated with 1,500 r survived the entire 8 days, in contrast to those which did not receive the preliminary irradiation—and of the rats receiving 3,000 r, those which had had preliminary irradiation, survived an average of 40 hours longer than those which had not.

The testes of the rats receiving preliminary irradiation showed virtually no damage from the single massive dose of 600 r (while those not previously irradiated showed extensive damage from this dose), and considerably less damage than in those not previously irradiated, from doses of 1,500 and 3,000 r.

The interpretation of these results is difficult and they cannot be translated directly into terms of radiation effect in man. Nevertheless, the results do point out the potential biologic significance of minimal roentgen doses.—*Henry G. Moehring, M.D.*

FINERTY, JOHN C., BINHAMMER, ROBERT T., and SCHNEIDER, MARTIN. Survival of irradiated rats in parabiosis with hypophysectomized partners. *Science*, Nov. 27, 1953, *118*, 654–655.

This experiment represents one of a series in which the mechanism of postirradiation protection by parabiosis is being sought.

Previous work has shown that neither the adrenals nor the spleen is necessary in the nonirradiated parabiotic partner to protect against irradiation.

In this experiment good protection was obtained when irradiated rats were paired with nonirradiated, hypophysectomized animals. This would tend to eliminate the role of the hypophysis and its dependent endocrine organs as critical factors in recovery from irradiation sickness.—*T. J. Wachowski, M.D.*

MÜLLER, K., and HASE, H. Die Bedeutung der Ultrafraktionierung für den biologischen Effekt bei Elektronenbestrahlung. (The biologic effect of ultrafractionation of electron radiation.) *Strahlentherapie*, 1953, *92*, 33–38.

Electron radiation from a weakly filtered radium source was rendered discontinuous by means of a stroboscopic disk. Eggs of *Drosophila melanogaster* were irradiated with the same total dose of continuous or interrupted electron and roentgen radiation so that half of the dosage was attained when the eggs were 4.5 hours old. The ratio of radiation to interruption in each cycle was 1:2. The rate of the interruptions was varied up to a maximum of 590 impulses of radiation per second.

Continuous radiation with an electron beam produced a higher rate of damage to the eggs than interrupted radiation. The damage rate rose with increased interruptions of radiation per second, but never reached the rate for continuous radiation. The greatest difference (19.5 per cent) between continuous and interrupted radiation was observed when the rate of interruptions was 5 per second.

On the other hand, discontinuous roentgen radiation (180 kv. with no filter) produced a higher rate of damage to the eggs than did continuous roentgen radiation.—*Henry G. Moehring, M.D.*

COLLETTE, J. M. Essais de lymphographie expérimentale; activité de la hyaluronidase. Attempts at experimental lymphography; activity of hyaluronidase.) *J. belge de radiol.*, 1953, *36*, 276–292.

The ear of the rabbit was injected with various radiopaque contrast media and the lymphatic system visualized. Addition of hyaluronidase at the site of the injection definitely helped to get better lymphograms by probably decreasing the interstitial pressure causing thus a slower diffusion of the radiopaque substance.

Any of the contrast media currently used for arteriography and phlebography can be employed for lymphography. Usually 1 to 2 cc. injected subcutaneously gave fine results provided the films were exposed at the right moment, this moment depending on the opaque media used.—*J. N. Sarian, M.D.*

COLLETTE, J. M. La microradiographie; bases et principes de la technique; une nouvelle application: la microlymphographie. (Microradiography; foundation and principles of the technique; a new application: microlymphography.) *J. belge de radiol.*, 1953, *36*, 293–312.

Microradiography consists in the realization and interpretation of radiological images of microscopic order. The microradiogram obtained should be either appropriate for microscopic examination or enlarged by means of microphotography. The clarity of the detail depends on the thickness of the preparation, hence histological techniques are used. From the radiographic point of view, the most important requirement is to use very soft rays, the time of exposure depending on the thickness of the histological section. Very fine grained emulsions are necessary. Another form of microradiography is based on the utilization of secondary electrons produced by impact of very hard rays (200 kv.) on a lead plate on which is placed the tissue covered by the radiographic film. Thus the penetrating rays traverse the film, the histological section and then hit the lead plate. It is

the secondary (rebounding) electrons which inversely traverse the section and imprint an image on the film.

Of the various instruments briefly described, the author has used the Machlett A_2 with an iron anode and a beryllium window. For one group of studies, in which iodine or at times thorium was used in the preparation of the section, the factors were 18 kv., 18 ma., focal distance of 35 cm. and one hour exposure. Good results were obtained in another group where greater detail was desired with the following factors: 2.5 cm. diameter, 14 cm. focal distance, 12.8 kv., 15 ma. and an exposure of 15 minutes. The characteristic rays of the iron anode appear to be satisfactory as to wavelength and penetration for these two groups of studies.

Using the above described techniques, the author successfully demonstrated the lymphatic channels and lymphatic valves of the ear of a rabbit after subcutaneous injection of a radiopaque medium. Nine very fine lymphograms are reproduced in the article. —*J. N. Sarian, M.D.*

Moos, W. S., Henderson, W. J., and Beattie, J. W. Various absorption coefficients for 23.5-Mev x-rays. *Science*, Sept. 11, 1953, *118*, 300–301.

The authors were interested in choosing the best material, with respect to lowest space consumption and cost as well as chemical stability, for a window through which patients receiving 23.5 mev. betatron roentgen therapy could be viewed safely. To achieve this it was necessary to measure the half-value layers of glass, lead glass, water and zinc bromide. For satisfactory protection for control room personnel it was found necessary to have a protective window thickness of about 8–9 half-value layers of absorbing material.

The technical set-up used for determining the absorption coefficients of the above mentioned materials with a 23.5 mev. roentgen beam is described in detail. The absorption curves obtained are shown on a graph; a table containing the half-value layers and absorption coefficients of the various materials is also included. Measured values for lead are given for comparison.

The authors note that the table shows only the rate of absorption due to ionization after the electronic equilibrium has been reached, which corresponds to a depth of approximately 4 cm. for water. The most efficient materials are in the following order: lead, lead glass, zinc bromide, glass and water.—*W. L. Somers, M.D.*

Mitchell, J. S., Smith, C. L., Allen-Williams, D. J., and Braams, R. Experience with the 30 MeV synchrotron as a radiotherapeutic instrument. I. Development, calibration and instrumentation. *Acta radiol.*, Oct., 1953, *40*, 419–434; II. Dosimetry and relevant physical measurements. *Acta radiol.*, Nov., 1953, *40*, 463–487; III. Approach to clinical use. *Acta radiol.*, Dec., 1953, *40*, 603–613.

A 30 mev. synchrotron provided by the British Medical Research Council was installed in November, 1949, at the Radiotherapeutic Centre, Addenbrooke's Hospital, Cambridge, England. Considerable time was spent in preparing this apparatus for clinical use and, as the authors state in the introduction, the problems of routinely operating it are still far from solved. They were able to treat only 5 patients between May, 1951 and March, 1952.

In the present article, which is divided into three main parts, an attempt is made to assess the value of this form of megavoltage roentgen therapy and to indicate its future possibilities.

I. The first part summarizes the development of the synchrotron preparatory to its clinical use and gives some details of construction, design and instrumentation required for its continuous operation. Particular attention is paid to the energy calibration, dose measurements and effect of the compensating filter, and to the beam localization, collimation and direction.

The disadvantages of the apparatus are the fixed roentgen-ray beam, the rather low dose rate and the noise produced by the magnet, although none proved to be very serious.

II. The second part deals with physical measurements relevant to practical dosimetry.

The roentgen rays emitted by the synchrotron are pulsed in character. The authors state that in their apparatus the roentgen radiation is produced in main pulses at the rate of 50 per second and that each main pulse, which is of about 10 microseconds' duration, consists of approximately 50,000 short pulses each of about 1×10^{-9} second duration. Employing the 100 r Victoreen chamber and using Boag's theory to calculate the collection efficiency they found, however, that this so-called ultrafractionation of the roentgen-ray beam presents no serious difficulty in dosimetry, although it probably must be taken into account in evaluating quantitatively the biologic effect on the tissues.

The dose is expressed in "r," great caution being exercised in relating this unit to the standard r unit. Values are given for axial depth doses with circular fields of 7.5, 5 and 2 cm. nominal diameters and isodose charts of depth dose distribution are included for circular fields of 9, 7.5, 5 and 2 cm. diameters. Also, information is furnished concerning measurements of backscatter, absorption coefficients and stray radiation.

III. The third part relates to the authors' clinical experience which, as already stated, extends to only 5 patients. The case histories of these patients are presented in detail to illustrate the method of procedure

as compared with the conventional methods used in 200 kv. roentgen therapy.

Most of the treatments were given with a dose rate of 8–11 "r" per minute measured in the filtered beam at 100 cm. focus-skin distance by using a single field technique.

The authors conclude by expressing their belief that 30 mev. roentgen therapy will prove useful in some malignant neoplasms which cannot be satisfactorily treated by other methods. They enumerate twelve such possibilities. At present, the main disadvantage of the 30 mev. synchrotron is its unreliability but it is expected that this difficulty will be overcome in the course of time.—*T. Leucutia, M.D.*

Lubert, Mortimer, and Krause, George R. Upright cholecystography using the fluoroscopic spot-filmer with graded compression. *Radiology*, Dec., 1953, *61*, 879–884.

The authors describe their technique for upright cholecystography using the fluoroscopic spot-filmer with graded compression. The compression cone flattens the gallbladder, thus reducing the density of its opacity whereby gallstones and other pathological conditions are seen through the opaque medium. The compression cone also assists in displacing gas-filled loops of the intestine from the gallbladder.

The advantages of this method over conventional methods are described. The spot-film exposures for average patients are made at 0.1 and 0.2 second using 67 to 75 kv. and 100 ma.

Roentgenographic reproductions demonstrating the increased accuracy of this method are included. —*Everett E. Seedorf, M.D.*

Kunkle, H. Melvin, and Carpenter, Earnest B. A simple technique for x-ray measurement of limb-length discrepancies. *J. Bone & Joint Surg.*, Jan., 1954, *36-A*, 152–154.

In this paper a method of limb-length measurement is described which is simple, accurate and requires a minimum of equipment. The equipment consists of any standard roentgenographic table with a Bucky grid, one sheet of lead-filled rubber 1/16 inch in thickness, and a few blocks of plywood, 3 inches wide by 6 inches long and of known thickness. The sheet of lead-filled rubber must accurately cover a cassette 14 by 17 inches. This sheet is then cut into three strips—one 8 inches wide and the other two strips each 4½ inches wide. The patient is centered on the roentgenographic table. The head of the table must be elevated approximately 20 to 25 degrees so that the body will press the patient's feet against the foot rest. It is essential that both feet should exert the same amount of pressure on the foot rest.

If both feet cannot be placed against the foot rest with the limbs extended, because of unequal leg lengths or deformities of the feet, this can be overcome by placing a sufficient number of plywood boards of known thickness beneath the foot on the short side, thus enabling both feet to exert the same amount of pressure, one against the foot rest and the other against its plywood supplement. A small sandbag is placed across the middle of the legs to prevent motion. A marker showing the total thickness of the plywood boards is put on the cassette for later reference.

The film cassette, 14 by 17 inches, must be placed square with the table to ensure accurate measurements when put in the Bucky tray. The 8 inch strip and one of the narrower strips of lead-filled rubber are then placed over the upper part of the cassette to mask it. The cassette, Bucky tray, and the tube are centered at the ankles. The tube must be at a true right angle to the cassette and at least at a distance of 30 inches to minimize distortion. An anteroposterior exposure is made of the ankle joints. The cassette carriage is then moved and centered under the knees with the 4½ inch strip of the lead-filled rubber moved down to the exposed lower portion of the film. In moving the tray, care must be exercised not to disturb the cassette in the tray. An anteroposterior exposure is made of the knees. The two smaller lead-filled rubber strips are placed over the lower portion of the cassette and the large one is removed. An exposure is then made of the hips after proper centering of the tube, and the film is ready for processing.

The processed film is placed on a view box and is measured by drawing horizontal lines perpendicular to the side of the film through identical points in the ankles, knees and hip joints (or in the pelvis in any case of hip-joint disease with destruction of the head of the femur or of the acetabulum). The vertical distance between the lines drawn through the ankle joints represents the difference in height of the feet. The difference between the lines drawn through the hip joints represents the total difference in length of the two extremities. If plywood blocks were placed under one foot, the height of these is added to the total length difference. The measurements on a metal ruler placed in the center of the roentgenographic table will be reproduced in the developed film. It is essential to fix the ruler lengthwise on the exterior center of the table with adhesive straps to keep it stationary. Accurate limb-length measurements can be made from the ruler.

The authors give two advantages of this method: The height of the feet can be measured and it also enables identical points in the pelvis to be used in total length measurements.—*Ralph S. Bromer, M.D.*

Radakovich, Michael, Dutton, Arthur M., and Schilling, John A. The effect of total body irradiation on wound closure. *Ann. Surg.*, Feb., 1954, *139*, 186–194.

The effect of total body irradiation on wound closure in rats was studied after administration of doses of 150, 450, and 650 r. Surgically produced wounds 2.5 cm. in diameter were found to heal as rapidly in those rats exposed to total body irradiation with the above doses as in nonirradiated controls.

In the animals receiving 650 r and surviving the lethal effect of the irradiation, a slight increase in the rate of wound closure was demonstrated.—*Walter T. Hileman, M.D.*

Lorenz, Egon, and Congdon, Charles C. Modification of lethal irradiation injury in mice by injection of homologous or heterologous bone. *J. Nat. Cancer Inst.*, Feb., 1954, *14*, 955–965.

Previous experiments have shown that injection of homologous or heterologous bone marrow protected mice against the lethal dose of roentgen radiation. The injection of marrow intravenously was always successful, while intraperitoneal injection was considerably less effective. The authors conducted experiments to determine whether bone implants might show a similar protective effect.

Three month old mice were given a single total body dose of 800–900 r. One to two hours later, the mice were injected intraperitoneally with the bone preparation. The bone was obtained from the humeri and femora of one month old mice and consisted of finely ground pieces of shaft devoid of marrow and adhering tissue suspended in buffered saline. Attempts at intravenous injection of the bone suspension failed due to the formation of emboli by agglutination of particles.

The control group of irradiated mice never survived beyond twenty-one days. In the injected mice that did not survive beyond the twenty-one day period, autopsy showed failure of the host's hematopoietic tissue to regenerate. In animals surviving beyond the twenty-one days (all surviving mice sacrificed 30–60 days following irradiation for histologic studies), the injected pieces of bone showed rearrangement of the bone spicules into small masses of bone with bone marrow formation in the marrow spaces. In these mice, the hematopoietic tissues of the host showed complete regeneration.

Similar experiments using heterologous rat bone demonstrated similar protection of the host against radiation but the transplanted bone did not form bone marrow.—*G. J. Di Bagno, M.D.*

Tahmisian, Theodore N., Passonneau, Janet V., and Adamson, Dorothy M. The effect of x-irradiation and lowered metabolic rate on the morphogenesis of developing *Melanoplus differentialis* embryos. *J. Nat. Cancer Inst.*, Feb., 1954, *14*, 941–953.

Regression in size occurs in diapause grasshopper embryos subjected to roentgen irradiation. Enzyme activity is increased, as manifested by a rise in hydroquinone oxidase. Practically every nucleus in the embryo becomes pyknotic, but the respiratory metabolism of the embryos undergoing regression is not inhibited.

The authors irradiated grasshopper embryos with a 15 ma., 200 kv. machine. Under aerobic conditions embryos exposed to 25,000 r underwent regression, while under anaerobiosis a dose of 240,000 r was required to produce this effect. The gas used for anaerobiosis was a mixture of 95 per cent N_2 and 5 per per cent CO_2. The metabolic rate apparently was markedly lowered by the gas. The caloric output was determined to be about 300 times lower under the gas than in the air.

Growth occurred, within limits, in the eggs irradiated at 2,500 r in air or 25,000 r under N_2/CO_2 while in diapause, placed in cold treatment to simulate winter, and then returned to developmental temperature at 25°C. During this postdiapause development their external morphology eventually resembled that of a twelve to fourteen day postdiapause embryo, but their development went no further. Chromosomal configurations were decidedly abnormal. Cell division continued to be abundant, but no tissue differentiation occurred, indicating that the capacity of tissues to differentiate physiologically is the process most sensitive to roentgen irradiation.

Eggs receiving 25,000 r in air or 240,000 r under N_2/CO_2 developed pyknotic nuclei by sixteen days of developmental temperature following irradiation, which resulted in regression in size. The regression is considered to be a result of catabolic activity, which persists after anabolism is stopped by roentgen irradiation. The authors suggest that this may have a bearing on the production of radiation-induced tumors. Roentgen radiation may inhibit anabolism and thus bring about regression of the tumor. At the same time, cells of normal tissue surrounding the irradiated tumor may be stimulated to divide, and thus cause *de novo* formation of malignant tissue. The capacity of tissue to continue differentiation or to redifferentiate is inhibited by irradiation; therefore, when normal tissue is sufficiently irradiated and begins cell division it may not be able to undergo induction into its own normal function, and a tumor may be formed.—*Arthur S. Tucker, M.D.*

Berglas, B., and Rubin, I. C. Study of the supportive structures of the uterus by levator myography. *Surg., Gynec. & Obst.*, Dec., 1953, *97*, 677–692.

In the cadaver, owing to loss of muscular tone, the anatomic relation between the uterus and the pelvic diaphragm is markedly altered.

The authors injected a "contrast medium containing half the usual amount of x-ray opaque substance contained in rayopake and without polyvinyl alcohol or any other thickening substance" into the pelvic

floor muscles of living subjects. The material did not cause pain, did not injure the tissues, and was eliminated by the kidneys within two hours. "With the patient on the examining table in lithotomy position, the levator crura on either side were palpated per vaginam. Under guidance of the palpating finger, a 21 gauge needle 1½ inches long was inserted through the lateral vaginal wall into the levator crus, and 5 cubic centimeters of the x-ray opaque substance injected on either side. Then the levator plate was palpated per rectum and its location determined. The needle was introduced into the levator plate in the midline and midway between the tip of the coccyx and the posterior circumference of the anus, and 10 cubic centimeters of the opaque substance injected into the levator plate." In addition, the vagina was visualized by filling it with 5 cc. of barium suspension or by insufflation with barium powder. The rectum was similarly filled with a barium suspension. The uterus was visualized by insertion of a metal stem-pessary into the uterine cavity. Roentgenograms were then obtained in various positions, including the upright, and with the patient relaxed and straining down.

Patients with pelvic organs in normal position and intact levator ani muscles were examined, and also patients with descent of pelvic organs and impaired function of levator ani muscles. It was seen that in the normal subject the levator plate is almost horizontal, the uterus lies over the plate, and with increased intra-abdominal pressure, is forced against the plate, not into the pelvic floor aperture.

When birth injury or some other structural or functional defect impairs the muscular pelvic diaphragm, the levator plate is depressed anteriorly, increasing the longitudinal diameter of the pelvic floor aperture. When the levator crura are impaired, the transverse diameter of the hiatus increases too. The result is that, with increased intra-abdominal pressure, the uterus is pushed down the inclined plane of the levator plate into the enlarged hiatus.—*George Cooper, Jr., M.D.*

Gustafson, Gordon E., and Cebul, Frank A. The effect of surgery on dogs following whole body x-irradiation. *Surg., Gynec. & Obst.*, Jan., 1954, *98*, 49–54.

This investigation was designed to determine whether or not surgical trauma superimposed upon whole body roentgen irradiation markedly altered the mortality.

Eighteen pairs of mongrel dogs were studied, having a mean body weight of 8.6 kilograms. These dogs were paired on the basis of age, body weight, and white blood cell count. Both dogs in a pair were irradiated, and then one of them was selected for surgery. Ten pair received 200 r and 8 pair received 300 r. No antibiotics were used.

The physical factors of irradiation were as follows: 220 kv., 15 ma.; target skin distance of 148.5 cm.; 1 mm. of aluminum and 0.5 mm. of copper filtration.

Twenty hours following irradiation a 10 cm. segment of ileum was removed approximately 15 cm. from the ileocecal valve. Laboratory study consisted of: white blood cell count, red blood cell count, hemoglobin determination, total serum protein value and body weight. The results were as follows:

(a) Mortality. Three of the 10 control dogs receiving 200 r died, a mortality of 30 per cent. Two of the 10 operative dogs receiving 200 r died, a mortality of 20 per cent. Of the 8 pair of dogs given 300 r, 4 controls died and 6 operative dogs died, giving a mortality rate of 50 and 75 per cent, respectively. The over-all mortality for the control dogs was 39 per cent, and for the operative dogs was 44 per cent. It is evident that the surgery did not alter the mortality in irradiated dogs. However, one cannot determine from this study what the results of surgery would be if it were delayed for a long period of time.

(b) Survival time. In the 300 r group the survival time was the same. For the 200 r control group it was an average of seventeen days, and for the 200 r operative group it was an average of thirty-seven days.

(c) The white blood cell count decreased in the irradiated dogs, but increased in those that were operated on.

(d) The red blood cell count was decreased in all groups.

(e) Hemoglobin was decreased in all groups.

(f) There was a slight decrease in body weight in all groups.

(g) Total serum proteins were slightly decreased.

(h) The most common organ involved was the lung; next in order was the kidney, heart, and gastrointestinal tract.—*John Watts, M.D.*

Eldred, Earl, and Trowbridge, William V. Radiation sickness in the monkey. *Radiology*, Jan., 1954, *62*, 65–73.

In this study, the authors used 37 monkeys (*Macaca mulata*) as test animals for total body irradiation. The subjects were rotated on an axis placed 100 cm. from a 250 kv. G. E. Maximar unit using 0.5 mm. Cu and 1 mm. Al filtration, half-value layer of 1.7 mm. Cu. The dose rate was 13.7 r/min. The test animals received single doses of 400, 550, 600, or 800 r.

The LD 50/30 days for the monkey was found to be approximately 600 r and LD 100/30 days close to 800 r. The higher the dose, the quicker the monkey died. Sex did not influence the length of survival or the morbidity.

The phases in the succession of physiological changes that follow near lethal irradiation are first vomiting and the lessened activity momentarily associated with it; then, as in man, after a short symptomless phase, a period of digestive symptoms begins manifested by anorexia, diarrhea and weak-

ness, especially severe in the heavily irradiated animals and may end in early death.

If the animal survives, this phase runs into one characterized by hemorrhages and infections. The total white blood cell count is now minimal and a second wave of death occurs.

Monkeys surviving these critical stages recover slowly. Some may still die over the next few months. Epilation is minimal and conspicuous hemorrhage is seen only in the 700 r dosage group. Alteration in the blood picture is the most sensitive index for lethal radiation exposure. The central nervous system escapes apparent damage.—*Dean Nichols, M.D.*

Conference on radiobiology and radiation protection, Stockholm, September 15–20, 1952. *Acta radiol.*, Jan., 1954, *41*, 1–108.

In the introductory note G. de Hevesy and R. Sievert state that a conference on radiation biology and radiation protection was held in Stockholm, Sweden, from September 15 to 20, 1952. Those invited to attend included members of the Joint Committee on Radiobiology, the International Commission on Radiological Protection and the International Commission on Radiological Units as well as a few selected lecturers and guests.

The two most important topics discussed at this conference were the following: (1) What doses per year and per capita of the population are, according to our present knowledge, permissible from a genetic point of view; and (2) What radiation doses, under different conditions, cause significant blood changes?

The inaugural address was delivered at the opening of the Conference by Arthur Thomson, Chancellor of the Swedish Universities. All the formal papers were presented on September 16. The remaining days were taken up by separate meetings of the three commissions and by joint sessions.

The eleven following abstracts are of the papers presented at the Conference.—*T. Leucutia, M.D.*

Muller, H. J. The manner of dependence of the "permissible dose" of radiation on the amount of genetic damage. *Acta radiol.*, Jan., 1954, *41*, 5–20.

Muller believes that the question of what dose of radiation should be regarded as permissible cannot be answered categorically. The objective of all actions is to do the most good with the least harm to everyone concerned, including posterity. This makes it mandatory to consider the long range genetic damage of radiant energy, as well as its immediate benefits. He states that each mutant gene received by an offspring will, on the average, constitute the deciding cause of what may be called the "genetic death" of one descendant—that is, of its dying before reaching reproductive age or failing to reproduce. Ordinarily, this will happen many generations after the mutant gene has arisen. In the interim, all of us carry a fairly high number of mutant genes, which hamper us and may be called our "genetic load." Each of these must be eliminated by becoming, some time, a deciding cause of genetic death on the part of some descendant, usually a remote one. The total vigor, resistance and general fitness of each person, apart from environmentally determined differences, depend on the combined actions of his genes.

It is estimated that about three "excess" mutant genes must be eliminated per eliminated individual in order to prevent an impossibly large "load" and an extinction of the population. Calculations indicate that there are at least three spontaneous mutations in ten human gametes or six in ten zygotes. Therefore 2 of 10 individuals in every generation suffer genetic deaths under normal conditions. The remainder have to reproduce sufficiently to offset this loss.

From Russell's data on mutations produced by roentgen rays in mice, it is calculated that 80 r would produce the same rate of mutation that occurs spontaneously, that is, would double the rate. If applied to man, this would require (1) the genetic death of 4 of every 10 individuals in each generation to establish an elimination equilibrium, and (2) a higher rate of reproduction to maintain the population. Muller believes that a 25 per cent increase in the mutation rate (produced by about 20 r) would approach the danger level for people living under primitive conditions of birth and death. Even less would be tolerated by those living in the modern way, that is, conditions of low birth rate and increased longevity.

Muller proposes the establishment of a per capita average ceiling of irradiation, beyond which it is biologically dangerous for the species as a whole to go. He decries the unwarranted exposure of the gonads to radiation for diagnostic or therapeutic purposes and urges the development of protective devices and of alternative non-radiation methods.—*T. J. Wachowski, M.D.*

Williams, E. K. The white cell count in relation to occupational radiation dosage. *Acta radiol.*, Jan., 1954, *41*, 21–29.

The author discusses the technical variation of the white blood cell count and the physiological variations of the individual's blood.

The coefficient of variation due to instrumental and field errors varies from 8 to 10 per cent. The evaluation of the different types of cells varies from 10 to 20 per cent. These technical errors are not as critical nor as great as the physiological variations that occur in the white cell content of the blood.

There is a diurnal variation in the total white blood cell count of normal individuals ranging up to 16 per cent. Exercise and emotional states cause a transitory leukocytosis in the peripheral blood. Initial counts of applicants for employment are higher

than subsequent counts probably due to the emotional stress of the initial blood sampling.

In a series of 3,098 pre-employment blood counts, 6 per cent of the leukocyte counts and 14 per cent of the lymphocyte counts were below the lower levels of normal given in the 1943 edition of the Recommendations of the British Roentgen-Ray and Radium Protection Committee. The individuals with the persistent low counts did not seem to have a greater risk from exposure to radiation than those with normal counts as judged by relative sickness rates.

Individuals subjected to accidental high exposure to radiation have an increased number of refractive, neutral-red staining granules in the lymphocytes. These persist for up to two years following exposure. Although the granules are not specific for radiation, they may prove a useful guide to exposure. Abnormal and degenerative changes in the white blood cells are also seen following excessive exposure.

The author believes that where good protection and monitoring exists greater efforts should be made on individuals exposed to higher risks or when positive results may be obtained, with a concomitant relaxation on "potentially exposed" personnel who are also monitored. Further study of cytologic alterations may prove to be a more sensitive index of radiation effect than gross numerical variation.—*Frank J. Rigos, M.D.*

MORGAN, KARL Z. Maximum permissible concentration of radioisotopes in food, water and air and maximum permissible equilibrium amounts in the body. *Acta radiol.*, Jan., 1954, *41*, 30–46.

This article presents in some detail the considerations involved in the establishment of maximum permissible amounts of radioisotopes in the human body, and maximum permissible concentrations in air and in water. The tables listing the values obtained will appear in the National Bureau of Standards Handbook 52. Table I provides a handy reference for users of radioisotopes to obtain accepted tolerance values for 81 isotopes, including those used in routine clinical practice. Table II lists maximum permissible concentrations in water under emergency conditions for six fission products.

The attempt has been made to evaluate each isotope in terms of energy of emission, relative biological effectiveness (dependent on specific ionization), biological half-life, and organ concentration, such that the most radiosensitive organ concerned shall not receive more than 0.3 rem (roentgen equivalent man) per week. Several values given are based on specific information, but the majority are established on the basis of the most accepted of the available assumptions. The general equation used to calculate the permissible body burden is:

$$\mathcal{Q} = \frac{2.6 \times 10^{-3}\, m\, W}{\Sigma\,(E\ \text{eff})\, f_2}$$

in which $\mathcal{Q}$ = μc. in total body, f_2 = fraction in critical organ, $\Sigma(E\ \text{eff})$ = total effective energy in mev., m = mass of critical organ in gm. and W = 0.3/RBE (relative biological effectiveness) rep per week. In all calculations, the Standard Man established at the Chalk River Conference of September, 1949, was used as a source of weights and metabolic constants.

Equations for maximum permissible concentration of a radioisotope in air and in water, and of noble gases are presented. These equations are based upon the best available knowledge of the continuous concentrations required to maintain in equilibrium that quantity ($\mathcal{Q}$) which will result in the maximal permissible dose of 0.3 rem/week to the most sensitive irradiated organ.

Equations for concentrations in air and water under emergency conditions, resulting from a nuclear accident or atomic bomb explosion, are also given. For this special case, a total summated dose of 25 rem is accepted as permissible. These equations differ from those above, not only in the higher dose allowable, but also in the assumption that assimilation is not continuous, but is confined to a definite time period with the activity both in the body and in the medium falling steadily.

The author considers the existence of a separate problem connected with radioactive particles, and discusses the dosimetry of small spherical sources of particulate radiations. Particles would only be a special hazard if the surrounding tissues receiving relatively high doses of radiation were to show a high incidence of subsequent tumor development.—*R. Kenneth Loeffler, M.D.*

BACQ, Z. M. The amines and particularly cysteamine as protectors against roentgen rays. *Acta radiol.*, Jan., 1954, *41*, 47–55.

The action of roentgen rays on aqueous solutions and on living organisms is due to the liberation of free radicals originating from the hydrolysis of water in the presence of oxygen. Certain substances can partially inhibit this action of roentgen rays on enzyme solutions and living organisms.

The author systematically studied the effects of amines and of substances bearing the—NH_2 function. The 2 sulfur bearing amines, cysteamine and cystamine were investigated extensively since the —SH function has a favorable protective action.

Twenty five hundred mice were used in the experiments. Control groups were employed with each investigated substance. The factors of irradiation were: 250 kv., 2.5 mm. Cu, 50 cm. distance, 100 sq. cm. field and 90 r per minute. Equimolecular doses of the substance tested (when toxicity permitted) were dissolved in 0.25 mil. of saline and injected intraperitoneally, 1 to 4 minutes before irradiation. A single dose of 700 r kills 98 per cent of the controls.

Cysteamine is a fragment of pantotheine which is a growth factor for *Lactobacillus bulgaricus* and a fragment of coenzyme A. It is not very toxic. If a

dose of 3 mg. of cysteamine per 20 gm. mouse is injected immediately before a dose of 700 r, 97 per cent of the mice survive. In order to obtain the same mortality curve as that of the controls, one must give at least 1,300 r. If the injection is made immediately after irradiation the mortality curve is identical with that of the controls.

Cystamine is at least as good as cysteamine; it liberates large amounts of histamine and is reduced to cysteamine in vitro by cysteine and also in vivo probably by glutathione and other—SH bearing protides. Recently it has been found that cystamine by mouth is an outstanding protector for mice, and that 800 mg. are well tolerated by man.

Cysteamine when injected is rapidly metabolized; it has disappeared from the blood of dogs and rabbits fifteen minutes after intravenous injection and has been excreted unchanged in the urine during the period. Its protective action (and also that of cystinamine) against roentgen rays is of short duration: one hour after the injection, the radiosensitivity of the mice has come back to normality.

Cysteamine also induces a long lasting (at least five hours) liberation into the blood (rabbit) and urinary excretion (dog and man) of reducing substances, one of which is ascorbic acid. This reaction is not the same as that of ACTH; it is not produced by cysteine. Cysteamine acts directly on cells. Both cysteamine and cystamine partially inhibit the oxidation of ferrous sulfate by roentgen rays. Cysteamine has marked antimitotic properties for muscle cells of chick embryo in tissue culture. Its action seems mainly on the protoplasm, not on the nucleus. In some cases of human chronic leukemia, cysteamine has given good results.

If rats are given cysteamine after a dose of 1,000 r with the liver protected, the mortality is reduced greater than if no cysteamine is given. There seems to be a curious synergy between liver protection and cysteamine action. This synergy might be explained by the fact that the liver is the main scource of ascorbic acid liberated by the drug injection and that ascorbic acid seems to play a role in the biochemical lesion resulting from roentgen irradiation.

Two hundred mg. of cysteamine injected intravenously at any time except one hour before irradiation is well tolerated and active against radiation sickness. Diarrhea, vomiting, nausea, and general weakness disappear after one or two injections. This drug does not decrease the beneficial effects of the radiation therapy and is now used in the Department of Radiotherapy of the University of Liège.—*Frank J. Rigos, M.D.*

Forssberg, A. Some remarks on chemical protection against ionizing radiation. *Acta radiol.*, Jan., 1954, *41*, 56–58.

The author in collaboration with N. Nybom investigated the protective effect of cysteine on *Allium* roots by correlating the growth rate and survival with cytologic characteristics and associated biochemical events.

The results of the investigation are given in detail. They agree fairly well with those of Gray, Read, Thoday, and others, obtained after irradiating *Vicia* roots in oxygen as well as in nitrogen, thus indicating a certain resemblance in the effect of cysteine and anaerobiosis.

Briefly, cysteine protects *Allium* roots from roentgen-ray damage by a factor of 2. The number of total chromosome disturbances (fragments, bridges) was reduced 50 per cent or more. The protection from alpha-ray damage, however, was much less pronounced, corresponding to a factor of 1.5 at the most. —*T. Leucutia, M.D.*

Bacq, Z. M. On chemical protection against ionizing radiation. *Acta radiol.*, Jan., 1954, *41*, 59–60.

The author gave 20 mice 700 r using 250 kv., 2.5 mm. Cu, 50 cm. distance, 100 sq. cm. field and 90 r per minute. Ten of these animals were given 3 mg. of cysteamine intraperitoneally before irradiation, and 10 served as controls.

During the first twenty-four hours following irradiation, the weight and the white blood cell count of both groups fall about the same degree. After forty-eight to seventy-two hours the loss of weight in the protected mice stops and then the weight starts to increase. In the controls, the weight continues to decrease until death. It seems that the presence of cysteamine during irradiation has protected some regeneration factor (possibly coenzyme A). The white cell count of irradiated protected mice begins to rise (on the 8th day if 550 r is given and on the 10th day if 700 r is given) earlier than in the controls when they survive.

There is no regeneration of the ovaries in either group and the adrenal cortex remains thin. Each tissue is affected by irradiation whether cysteamine is present or not. It is the regeneration and the reactions of the organism against radiation damage which are enhanced when cysteamine is present.—*Frank J. Rigos, M.D.*

Latarjet, R., and Gray, L. H. Definition of the terms "protection" and "restoration." *Acta radiol.*, Jan., 1954, *41*, 61–62.

The authors distinguish the following four stages in the development of any particular change in a cell (tissue, organ or organism), resulting from exposure to radiation, which they call the "observable lesion:"

1	2	3	4
Absorption of radiant energy	→ Primary radiochemical reactions	→ Chemical reaction chains	→ Observable lesion
	latent period		

In the first stage, the distribution of the absorbed energy within the cell is determined by the chemical structure of the cell and the type of radiation to which it is exposed. The absorbed energy will be distributed within 10^{-13} seconds. This is followed by the second stage, in which part of the energy is in the form of very reactive and very short-lived radicals and which is probably complete in 10^{-9} seconds. In the third stage, the short-lived radicals give rise to intermediates of longer life and to reversible and irreversible chemical changes. Stages 2 and 3 together constitute the latent period during which the course of the observable changes is affected by the chemical composition of the living protoplasm. The changes in Stage 3 are further influenced by environmental factors such as temperature and nutrients. Stage 4 comprises the observable lesion.

The authors propose that the term "protection" be used to describe a diminution of the observable lesion resulting from a treatment or procedure instituted *before* the beginning of irradiation, and the term "restoration" when this is achieved *after* the beginning of irradiation. In this manner, the term restoration includes such procedures as photoreactivation and heat-reversal and also covers control of the natural process of recovery. It is emphasized that both protection and restoration refer to a particular observable lesion and not to the cell as a whole. For example, mutations may persist in a cell in which the ability to divide has been restored.—*T. Leucutia, M.D.*

GRAY, L. H. Conditions which affect the biologic damage resulting from exposure to ionizing radiation. *Acta radiol.*, Jan., 1954, *41*, 63–83.

At present, it is not possible to influence significantly the physical effects of a given type of radiation. The absorbed energy is converted within a fraction of a second into chemical reactions. During the conversion period, very reactive short-lived radicals are present, which give rise to intermediates of longer life and to reversible and irreversible chemical changes. During the conversion period, some control can be exerted over the development of a radiobiological lesion by modifying the enviromental conditions.

An overriding factor which determines the reactions to which the short-lived radicals give rise is the disposition of these radicals along the ionization tracks. If they are formed very close together, they react among themselves in a manner quite differently from when they are more sparsely distributed, and this profoundly affects the chemical and biological changes which follow. No chemical treatment at the time of irradiation, which has been tried so far, has been found to modify appreciably damage induced by the high ion density irradiations, such as from alpha and neutron beams.

Hypoxia or anoxia reduces the general damaging biological effect of many diverse types of radiations, if present during or immediately before irradiation. The effect on roentgen irradiation is a numerical reduction in the efficicacy of a given dose, irrespective of the magnitude of the dose. The mechanism is probably a reduction in the number of effective chemical intermediates produced per unit expenditure of energy of the secondary electrons generated by the radiation. Five different biological examples of the influences of oxygen tension on general radiosensitivity are cited.

While the effect of anoxia on chromosome damage of germinal and somatic cells is established, the effect on the other forms of genetic damage is not so clear. Further work in this field is needed.

Chemicals may alter the radiation effect by reducing the oxygen tension of the media, inhibiting respiration or increasing metabolism in the presence of a limited oxygen supply. The reducing sulfur compounds and strongly reducing amino acids are effective in this manner. The latter exert greater protection if present in the cell for about an hour before as well as during irradiation. They also can reduce radiation damage if administered shortly after irradiation.

Exposure to increased pressures of carbon monoxide for about an hour prior to and during roentgen irradiation increases chromosomal damage and inhibits the growth of roots. No significant increase is noted under similar conditions with neutron irradiation.

Because the infinitesimal amount of energy deposited in the cell by irradiation can do no more than initiate a sequence of changes which develop into a manifest lesion, the postirradiation period is a critical one and it is logical to attempt at that time to check the development of injury. Purely physical agents may either diminish or augment the ultimate damage resulting from irradiation. Conditions of incubation following irradiation influence the effect because of changes in the environment produced by the irradiation, such as in the media.

The effect of irradiation on animals, irradiated either wholly or in part, suggests interaction, such as between an injured organ and the capability for restoration latent in the organism as a whole, or as between one organ and another.—*T. J. Wachowski, M.D.*

LATARJET, RAYMOND. Spontaneous and induced cell restorations after treatments with ionizing and non-ionizing radiations. *Acta radiol.*, Jan., 1954, *41*, 84–100.

The recovery of cells from injuries produced by radiations is explained by three general processes which are illustrated by experimental effects resulting from superimposed temperature changes, chem-

icals (catalase) or visible light. Examples are quoted of the restoration of lesions caused by roentgen, α and β rays and infra-red but mainly refer to the effects of ultraviolet rays.

Three types of restoration processes are recognized: (a) Reversion, in which the reaction induced by the radiation is reversed or there is destruction of intermediate active compounds necessary for the ultimate production of a lesion. (b) Recovery, where the lesion is due to the inactivation or destruction of a cellular constituent, may be induced by reintroducing this constituent either by reviving its synthesis or from an external supply. (c) Superimposed injury, when the chain of reactions leading to the production of the lesion requires the presence of a normal cell metabolite, may remove this constituent and prevent the development of the lesion.

Spontaneous recovery of *Escherichia coli* strain B after ultraviolet irradiation shows that growth restoration is greater by a factor of 7 for cell clumps and appears to be dependent on close contact between the cells indicating that induction is due to the transmission of some growth restoring substance. In the case of yeasts, such natural restoration is dependent on diploidy.

Experimental evidence is considered for restorations produced by three factors: (a) Temperature change. Recovery may be induced by the destruction of activated states at high temperatures so cancelling the primary effect of the radiation by reversal or physiologic recovery may follow the differential effect of temperature on the speeds of intracellular reactions; this will, of course, work in both directions (heating or cooling), and occurs with ionizing or non-ionizing radiations. The complexity of the process is illustrated by the mutation of *Esch. coli* strain B to the radioresistant strain B/r: between 37° C. their relative resistances to radiation change from 1 : 10 to 1 : 1, that of B having increased 20 times for a two-fold increase in B/r. (b) Chemical reactions. The only unambiguous evidence of chemical restortion appears to be the effect of catalase on certain micro-organisms and does not occur after roentgen irradiation. The relation between the enzymatic activity of catalase and its restoring capacity suggests that this is due to the decomposition of peroxides, probably of the RO-OH type as H_2O_2 provides protection but not restoration. A proposed scheme to account for the restoration process is illustrated; it is pointed out that the proportion of cells restored by catalase is only about 1 in 100. (c) Light. Recovery from restorable lesions due to ultraviolet light in the range 3,500–5,000 Å. is related to N.P. synthesis by transforming inactive into active N.P. Generally, this is a rare phenomenon and is only successful after mild inactivation but in the case of roentgen irradiation of *B. megatherium* illumination at an early stage does not reverse the primary effects but prevents the completion of the induced process, the synthesis of active virus and cell lysis (superimposed lesion).—*Raymond Quick, B.S.*

Ehrenberg, L., and Gustafsson, Å. The effects of ionizing radiations in barley. *Acta radiol.*, Jan., 1954, *41*, 101–104.

This short general summary refers mainly to some recent studies in the production of useful mutants in agricultural plants and the implications of the experimental findings. It is pointed out that certain of these plants, such as barley, provide very suitable material for the investigation of many aspects of the early, intermediate and later effects of radiation.

In the case of dormant embryos, although the water content is low (10 per cent), hydrogen sulfide has a marked protective action, probably similar to that of sulfhydryl compounds in aqueous tissue. Any effect of oxygen pressure is small and is only observed for pressure changes twenty-four hours previous to irradiation indicating that either the penetration or the modification of the physiologic state is slow.

Germinating seeds are very sensitive to changes in oxygen pressure thirty minutes before irradiation. Radiation damage is increased by pure oxygen but nitrogen and hydrogen provide protection. Anesthetics generally protect against radiation (propene both for dormant and germinating seeds) but low ether concentrations increase damage.

With fast (3 mev.) neutrons, H_2S has no protective effect in dormant seeds and there is no influence of oxygen pressure on germinating seeds. Roentgen irradiation is followed by immediate death or recovery, but after neutron treatment, germination and a few days of normal growth may be terminated by sudden death and in mature plants, the spike fertility is much lower.

Lethal and semi-lethal mutations which are easily induced by ionizing radiations have highly complex viability characteristics, each mutation following a sequence depending on the environment and genetic conditions, but in barley, only 1 or 2 per 1,000 are completely viable. Chromosome breakage increases with densely ionizing radiations and leads to effects (2 point breakages) not observed spontaneously or with roentgen rays.

Increased exposure to ionizing radiation would be expected to lead to corresponding deleterious changes in man, such as long chromosome deficiencies, inversions and translocations, especially as the high percentage of damage cannot be eliminated as in the case of plants.—*Raymond Quick, B.S.*

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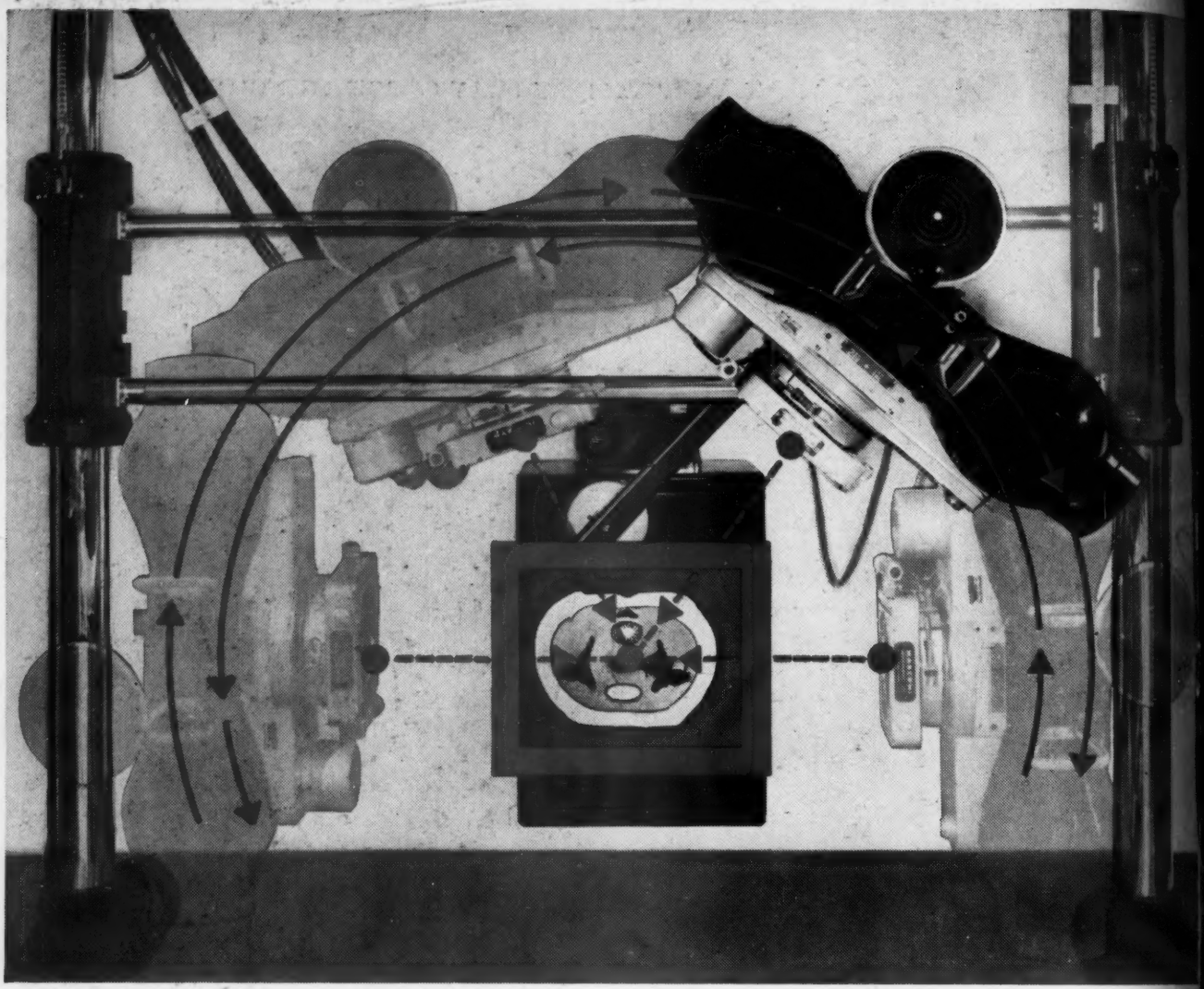

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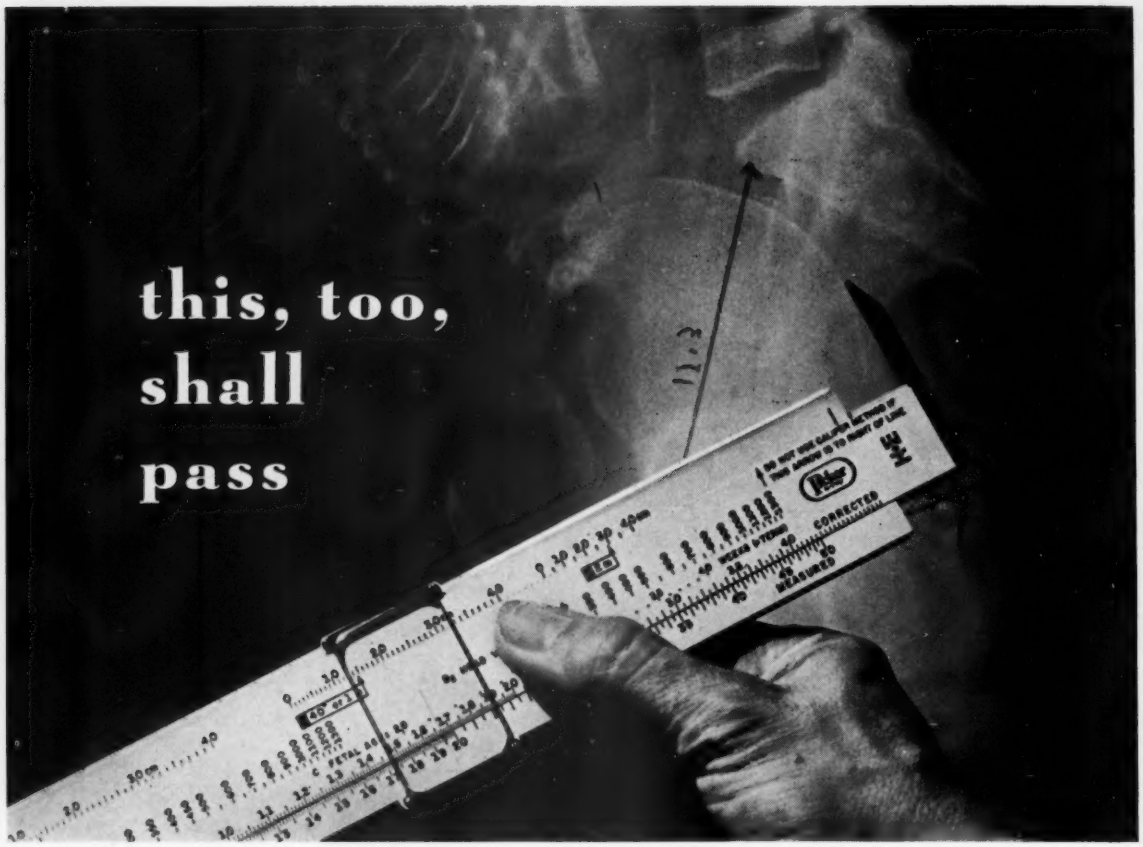

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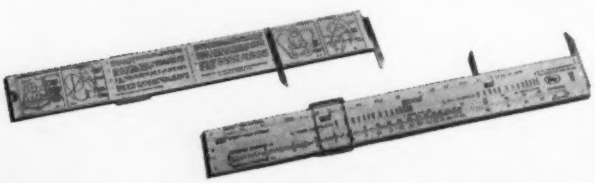

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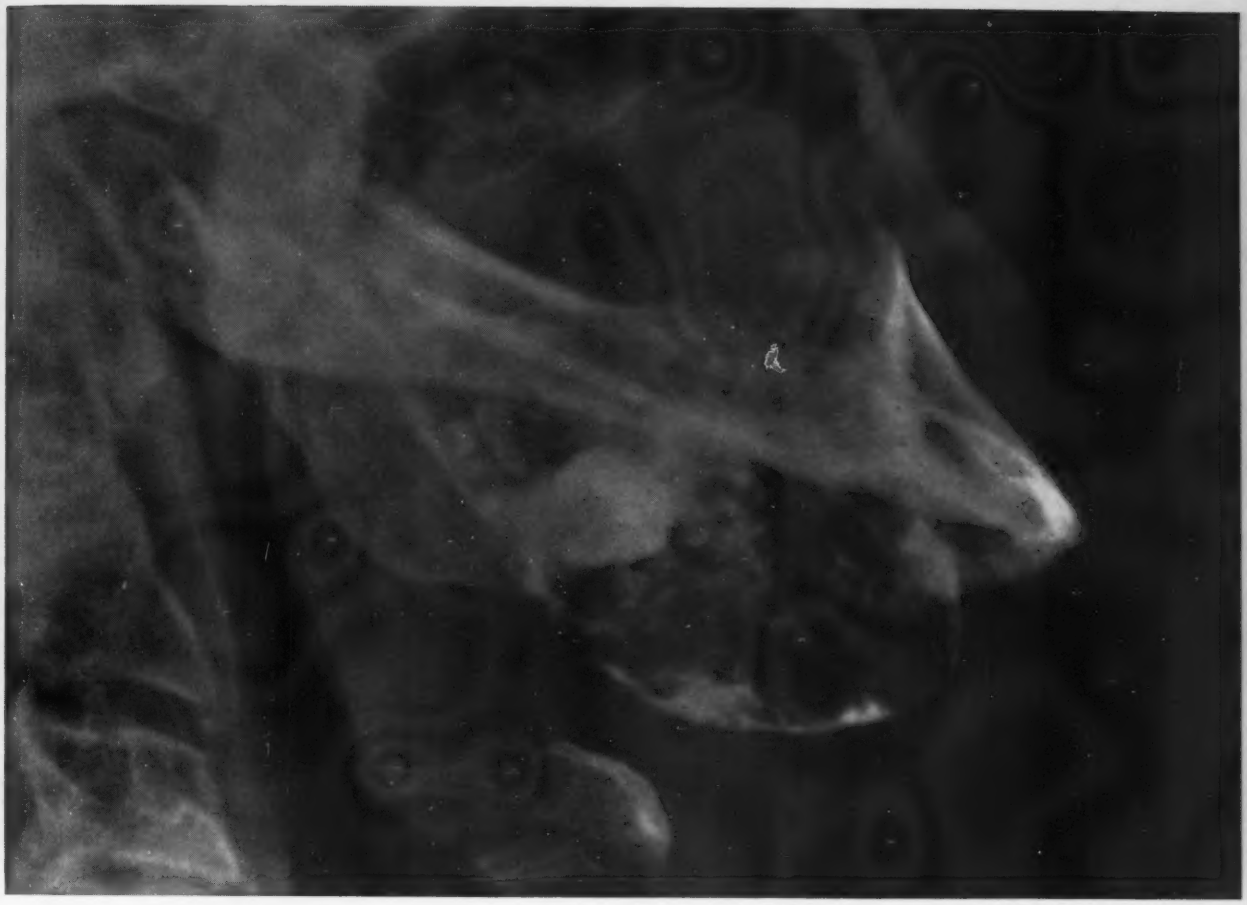

Preoperative lateral radiograph of the left mandible.

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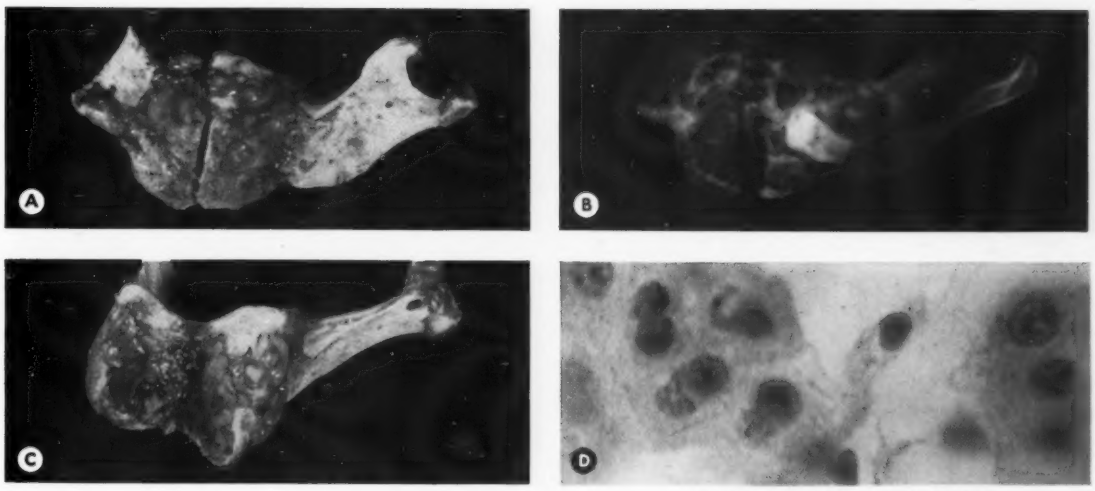

A. Photograph of resected mandible.

B. Radiograph of resected mandible.

C. Photograph of resected mandible cut to show appearance of cross section.

D. Photomicrograph (×1000) of resected mandible.

The radiographs and color photographs reproduced on this and the following page illustrate a mandibular resection. Better than hundreds of words, they show how radiography and photography work, day in and day out, to aid physicians and students in diagnosis, teaching, and research.

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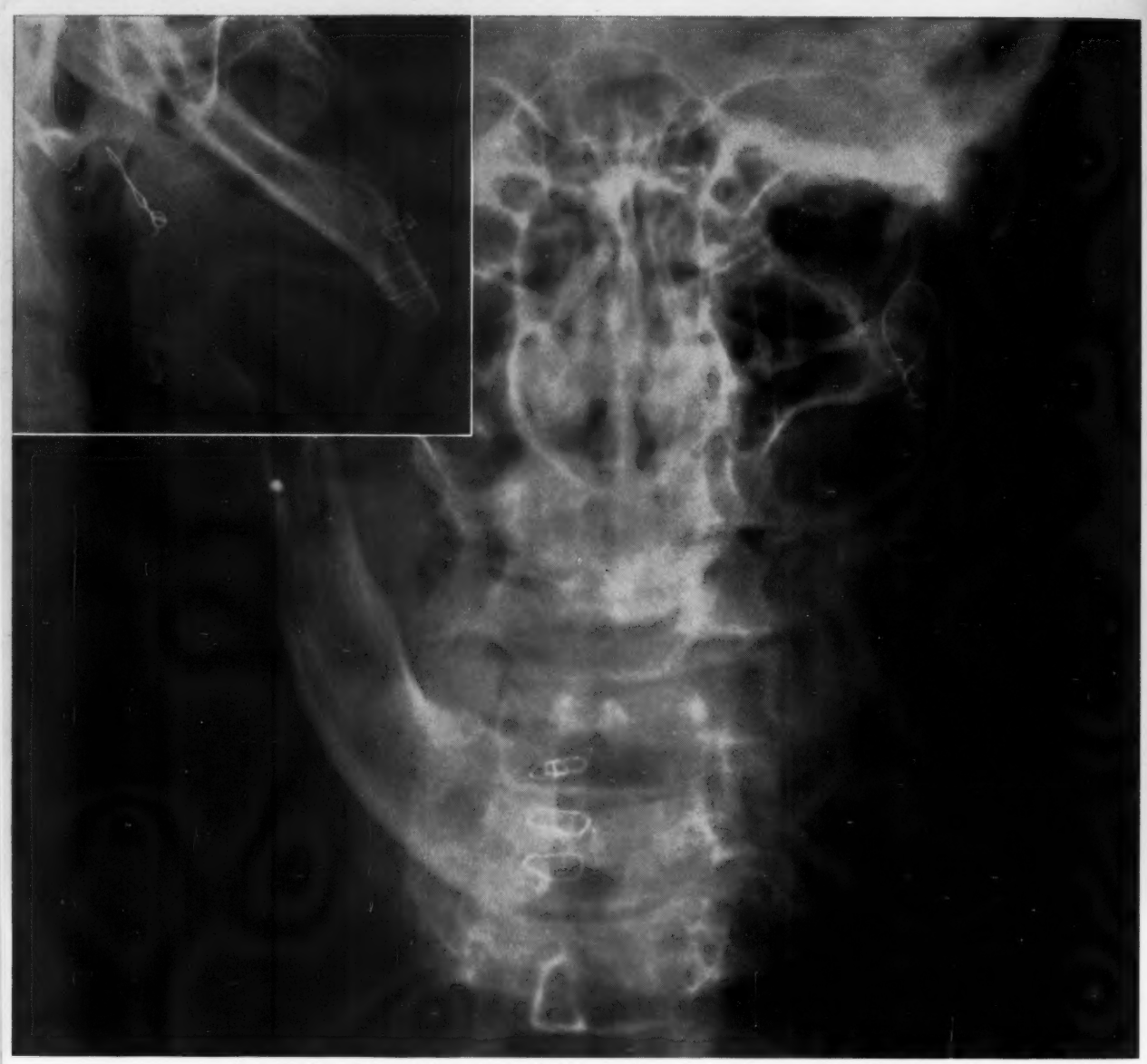

Postoperative posteroanterior and lateral oblique radiographs showing restoration of mandible with acrylic (radiolucent).

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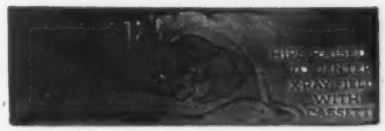

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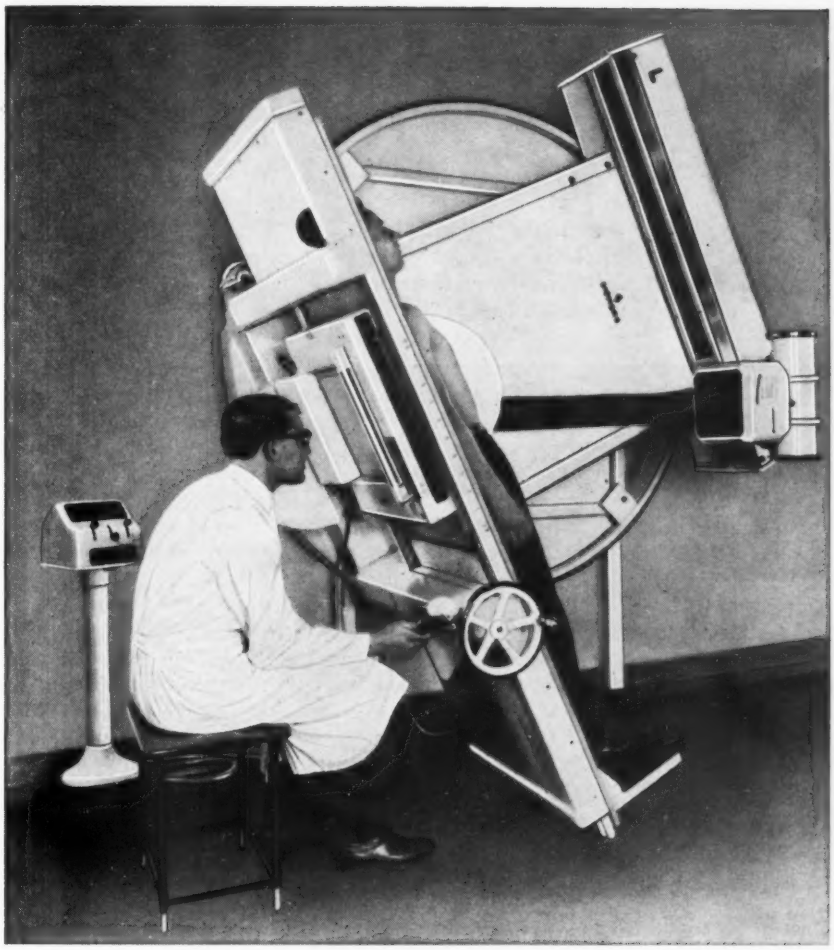

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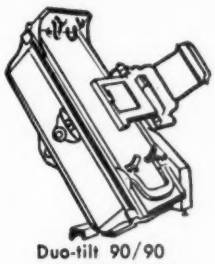

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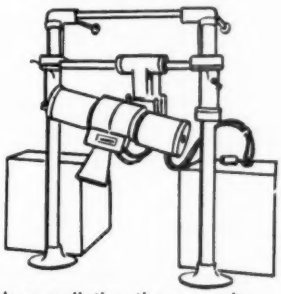

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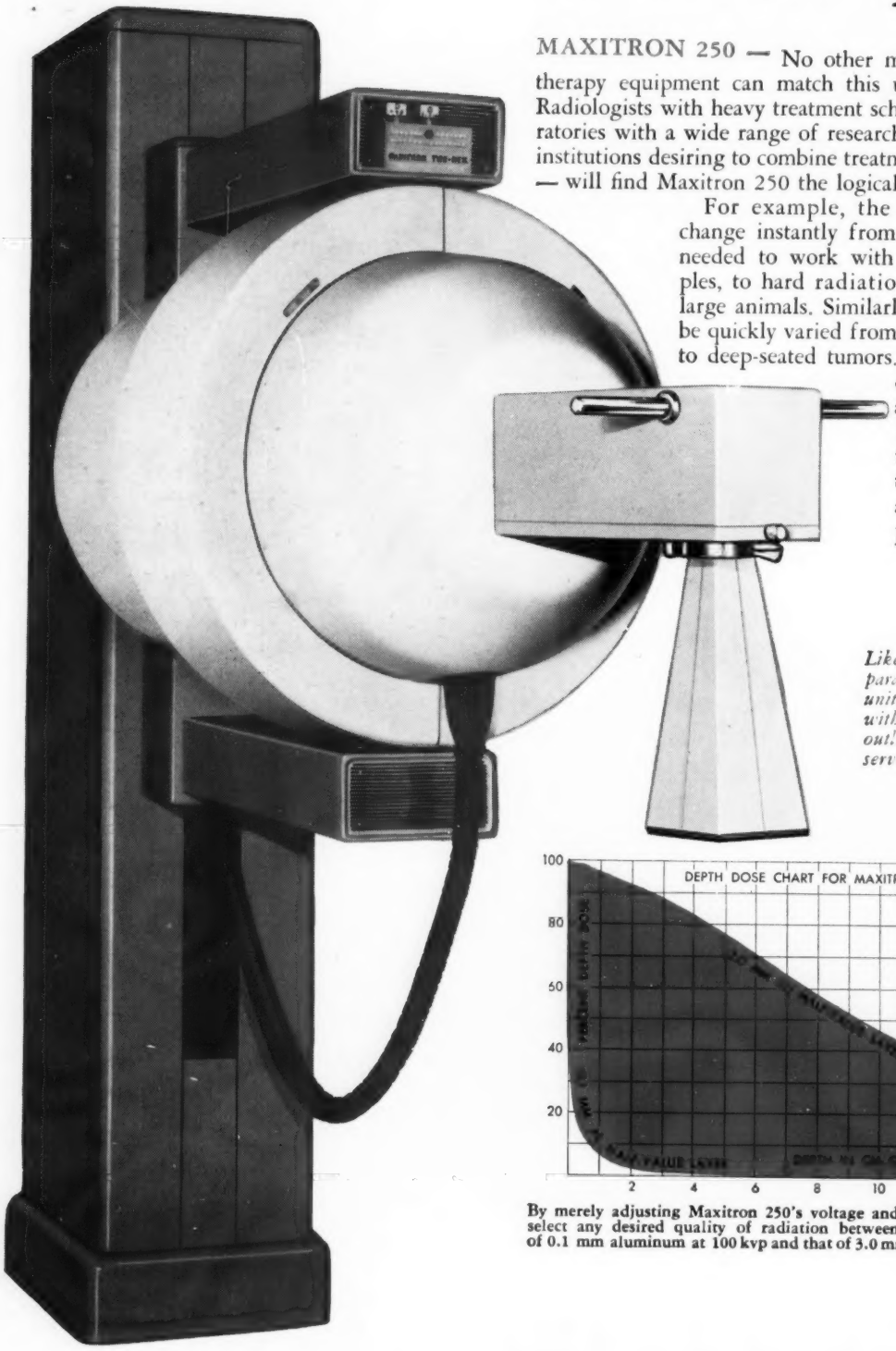

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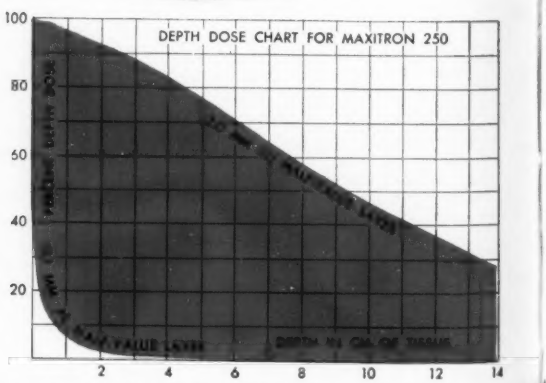

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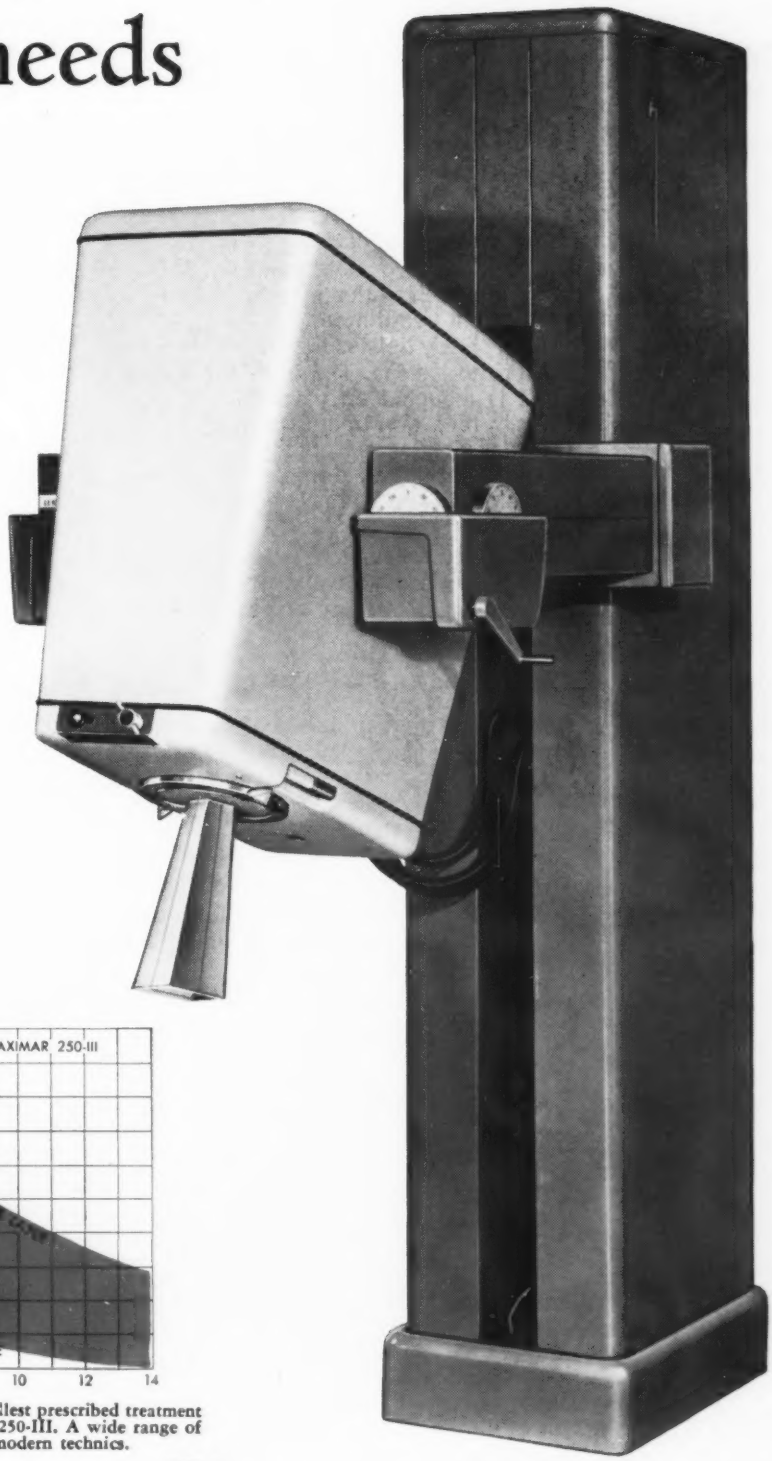

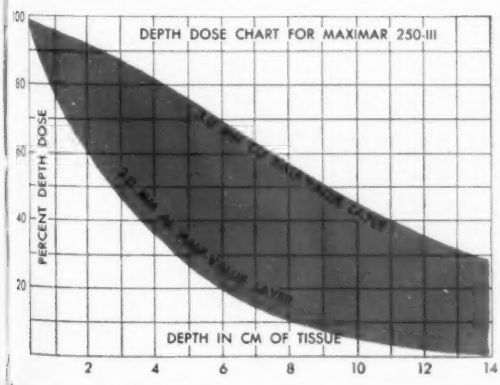

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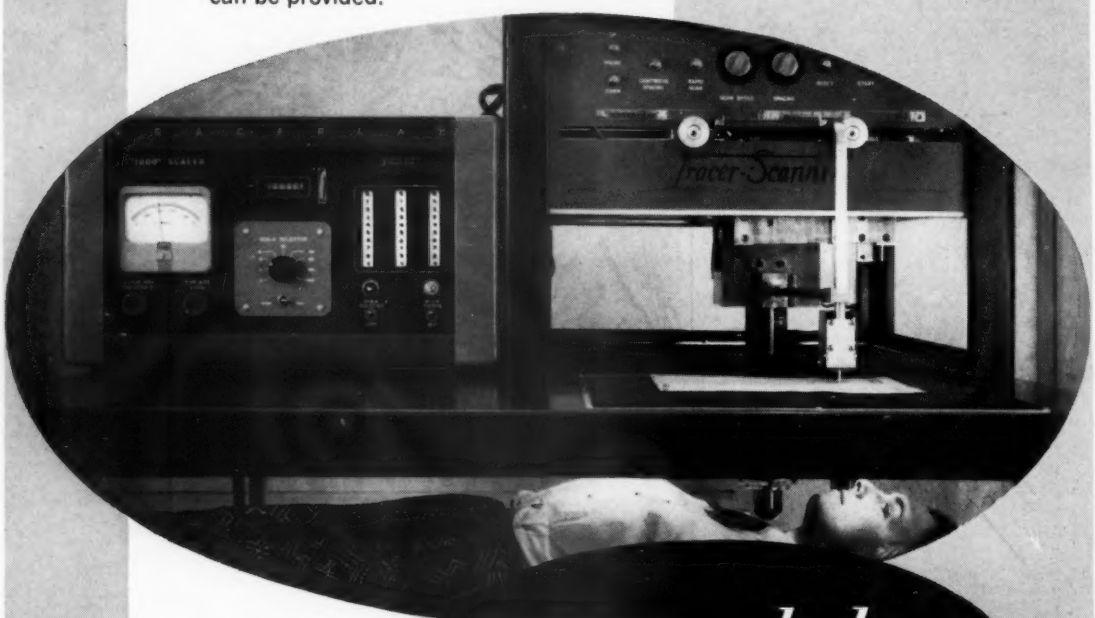

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Many investigators observed that UROKON is excreted more rapidly than other contrast media. The advantages of this are that *a pyelographic study may be completed in a shorter time with Urokon,* and if obstruction is present it is demonstrated more readily.[3]

[1] Porporis, A. A., Zink, O. C., Wilson, H. M., Barry, C. N., Royce, R., and Rose, D. K.: Radiology *60:* 675 (1953).

[2] Nesbit, R. M. and Nesbitt, T. E.: Univ. of Mich. Med. Bull., *18:* 225 (1952).

[3] Richardson, J. F. and Rose, D. K.: J. Urol., *63:* 1113 (1950).

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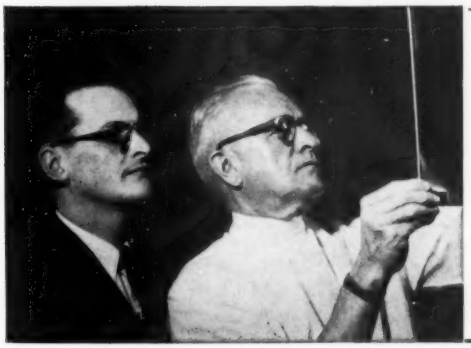

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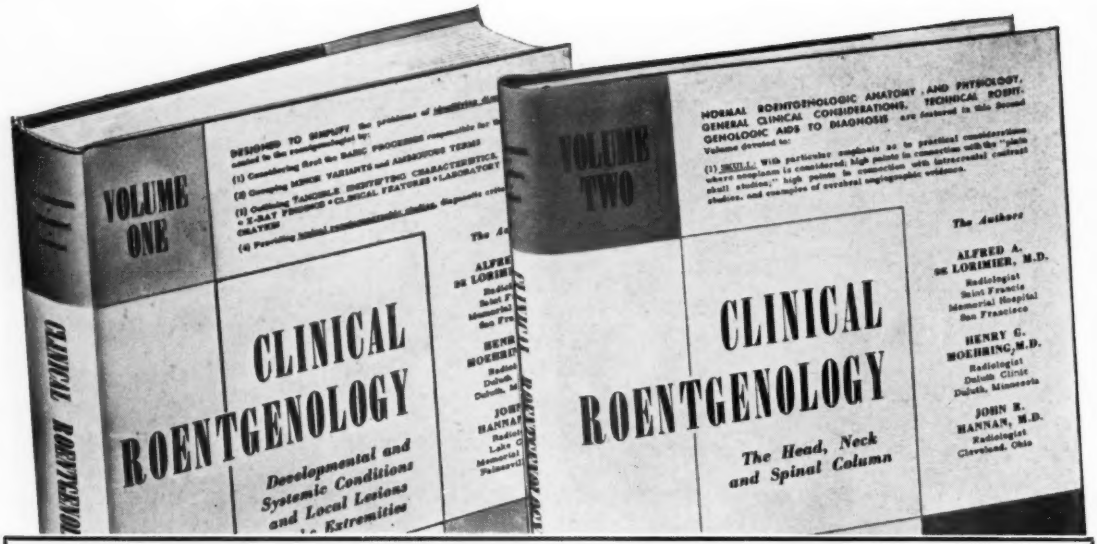

CLINICAL ROENTGENOLOGY

By

ALFRED A. de LORIMIER M.D.

Radiologist, Saint Francis Memorial Hospital San Francisco

HENRY G. MOEHRING, M.D.

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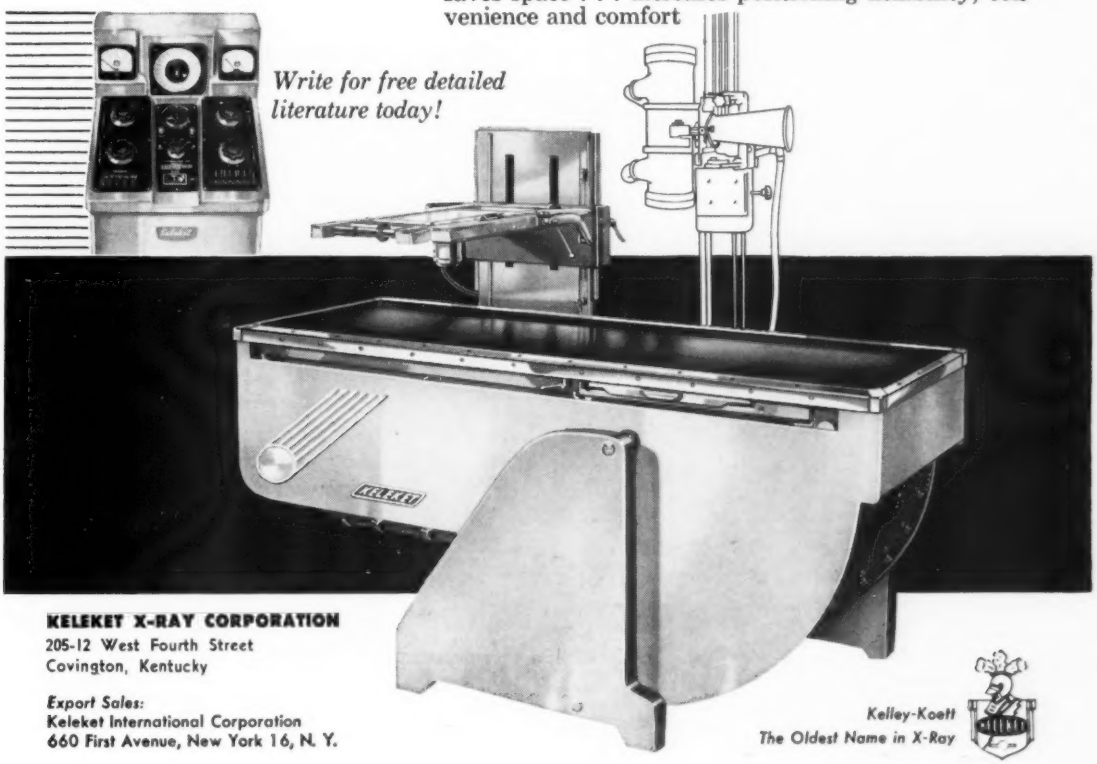

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FROM THE INTRODUCTION:

The research worker and the clinician often have neither time nor opportunity to read the literature dealing with subjects not directly related to their work, and they will be all the more apt to lose sight of the results of the latest investigations, as the relevant literature is scattered in a great number of different periodicals - often written in foreign languages. This applies above all to the field of antibiotics and chemotherapy, and it was therefore decided to meet the urgent need for collective reviews in the form of annual surveys reporting the latest results of research in this field. The world literature will be critically reviewed and referred to in detailed bibliographies. Thus the surveys will enable the specialist to keep up to date in his own field, at the same time supplying a review of the latest developments to all those not so well versed in this special field. The reports will further save the research worker the trouble of obtaining the original literature and enable all other scientists to overcome the difficulties of making themselves familiar with this particular field of medicine. With this aim in view, the annual survey will help to maintain contact between scientific research, clinic and daily practice.

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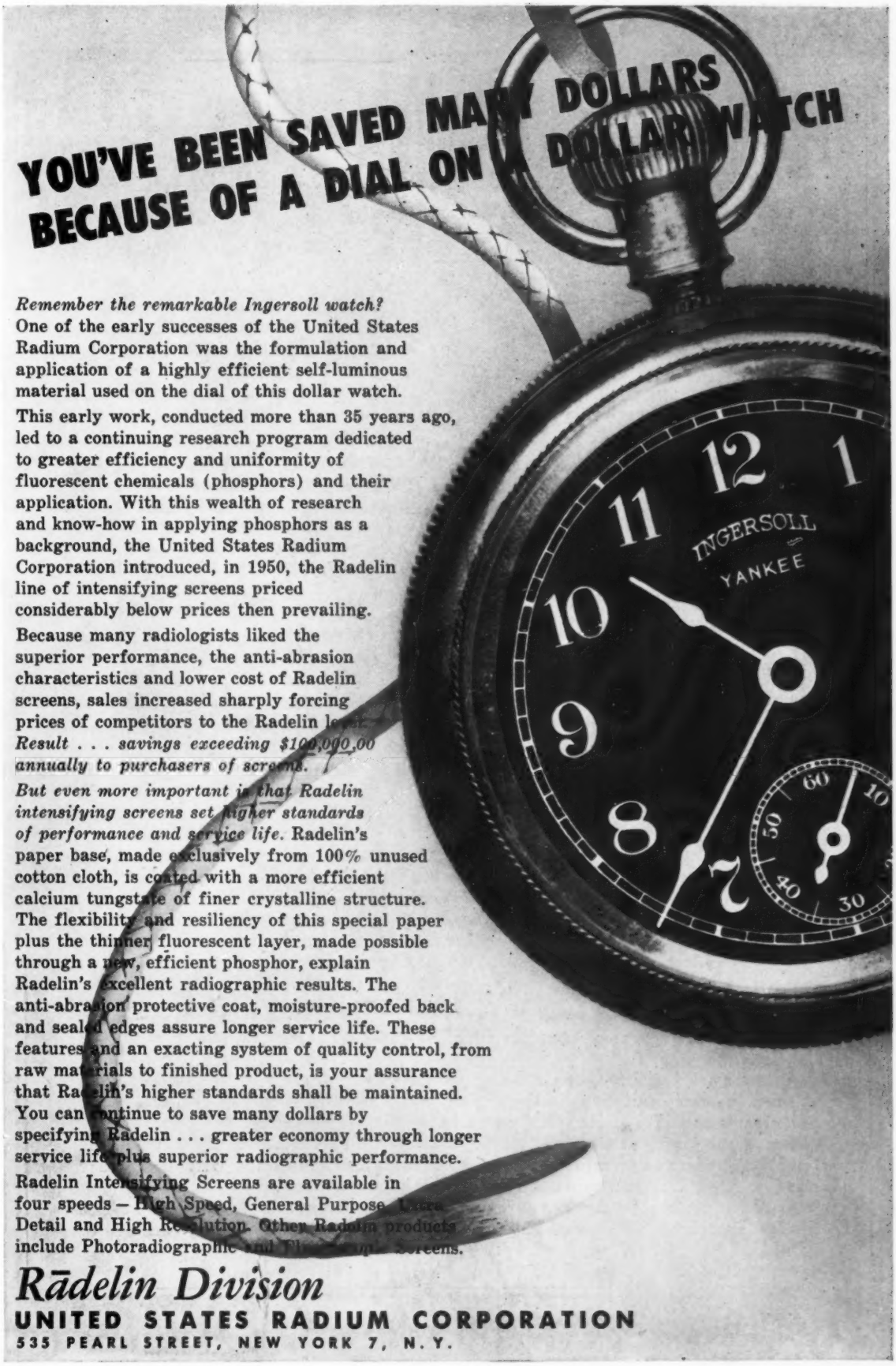

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Because many radiologists liked the superior performance, the anti-abrasion characteristics and lower cost of Radelin screens, sales increased sharply forcing prices of competitors to the Radelin level. *Result . . . savings exceeding $100,000.00 annually to purchasers of screens.*

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MEMORANDUM ON THE FIFTH INTER-AMERICAN CONGRESS OF RADIOLOGY

SHOREHAM HOTEL, WASHINGTON, D.C., APRIL 24-29, 1955

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Eugene Pendergrass, *Secretary-General*
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The Inter-American Congresses of Radiology have been held under the auspices of the Inter-American College of Radiology. Previous Congresses took place in Buenos Aires, Havana, Santiago (Chile) and Mexico City. This Fifth Congress is sponsored by the American College of Radiology representing all radiology of the United States of America.

THE PROGRAM OF THE CONGRESS WILL INCLUDE THE FOLLOWING:

1. **Forty communications** covering radiodiagnosis, radiotherapy, radiophysics and radiobiology.
2. There will be **three symposia** on (a) cancer of the lung (b) new trends in cancer and (c) radioactive isotopes.
3. **Refresher courses** (cursos de Repaso). This will be given in English or Spanish by outstanding authorities in different fields of the specialty; each course will last at least one hour and a half.
4. **Scientific exhibits.** This will be a visual display of the new contributions and accomplishment in radiology.
5. **Technical exhibits.** All manufacturers will display equipment and technical development of importance to the specialty.
6. On Saturday, April 30, one of the sessions of the Congress will be held at the *National Cancer Institute of the National Institute of Health* in Bethesda, Maryland, in the vicinity of Washington. The *American Radium Society* will hold its annual meeting in the Shoreham Hotel on April 21st to the 23rd, 1955, immediately preceding the Congress.

MEMBERSHIP in the Congress is open to **all doctors** of Medicine specializing in *radiology, radiophysics* and *radiobiology*. Privilege of attending is open to all doctors of medicine. An album of photographs of all those registering will be presented at the Congress.

An extensive *social program* is being planned to permit the visit to *medical* as well as other *cultural* institutions in the City of Washington. This will be available to the participants as well as their social associates.

One evening will be devoted to a private concert by the Philadelphia Symphony Orchestra under the direction of Eugene Ormandy in Constitution Hall, Wednesday evening, April 27. Numerous tours and special visits are planned.

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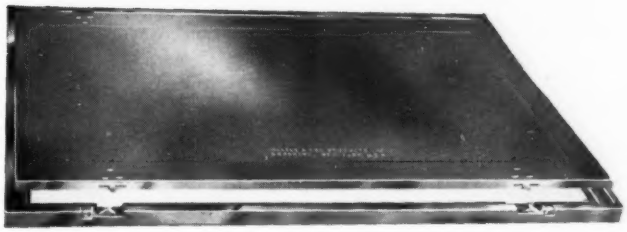

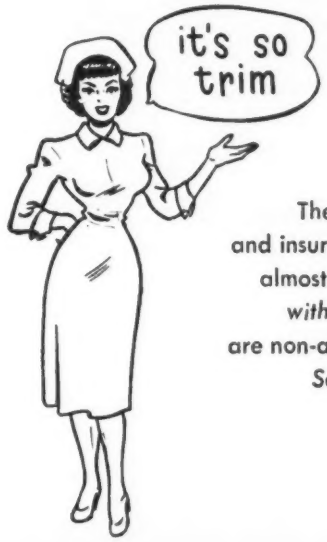

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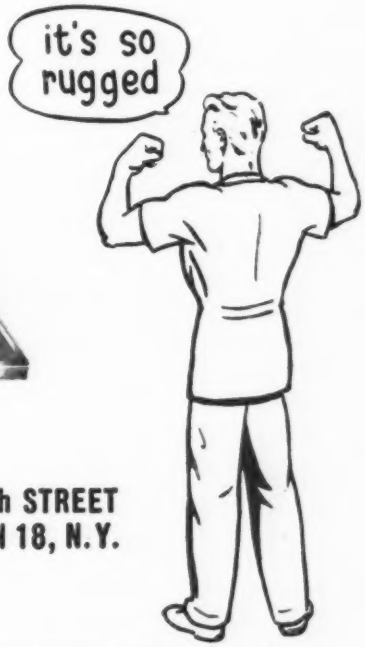

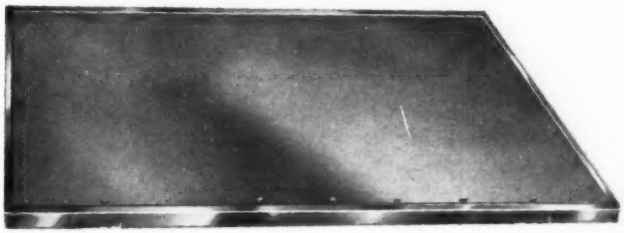

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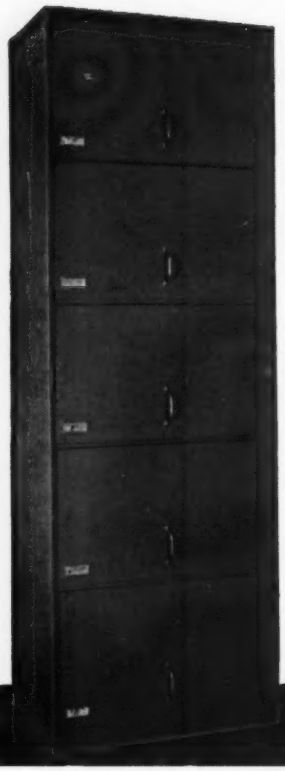

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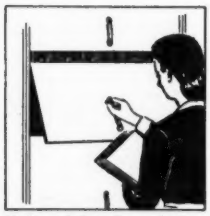

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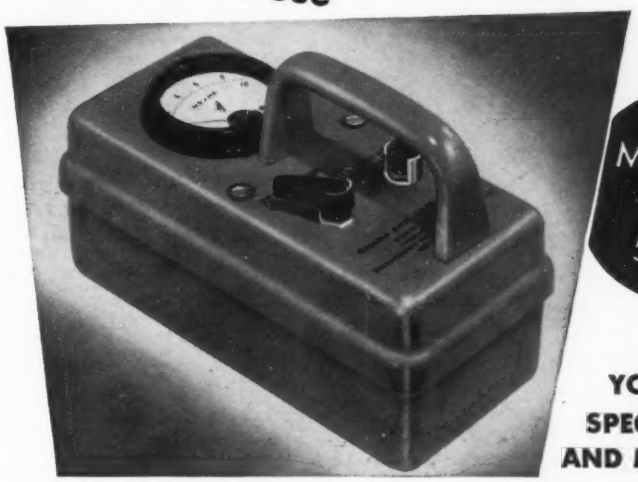

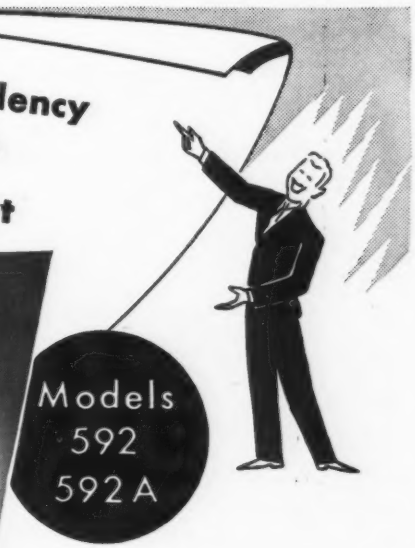

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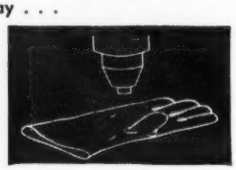

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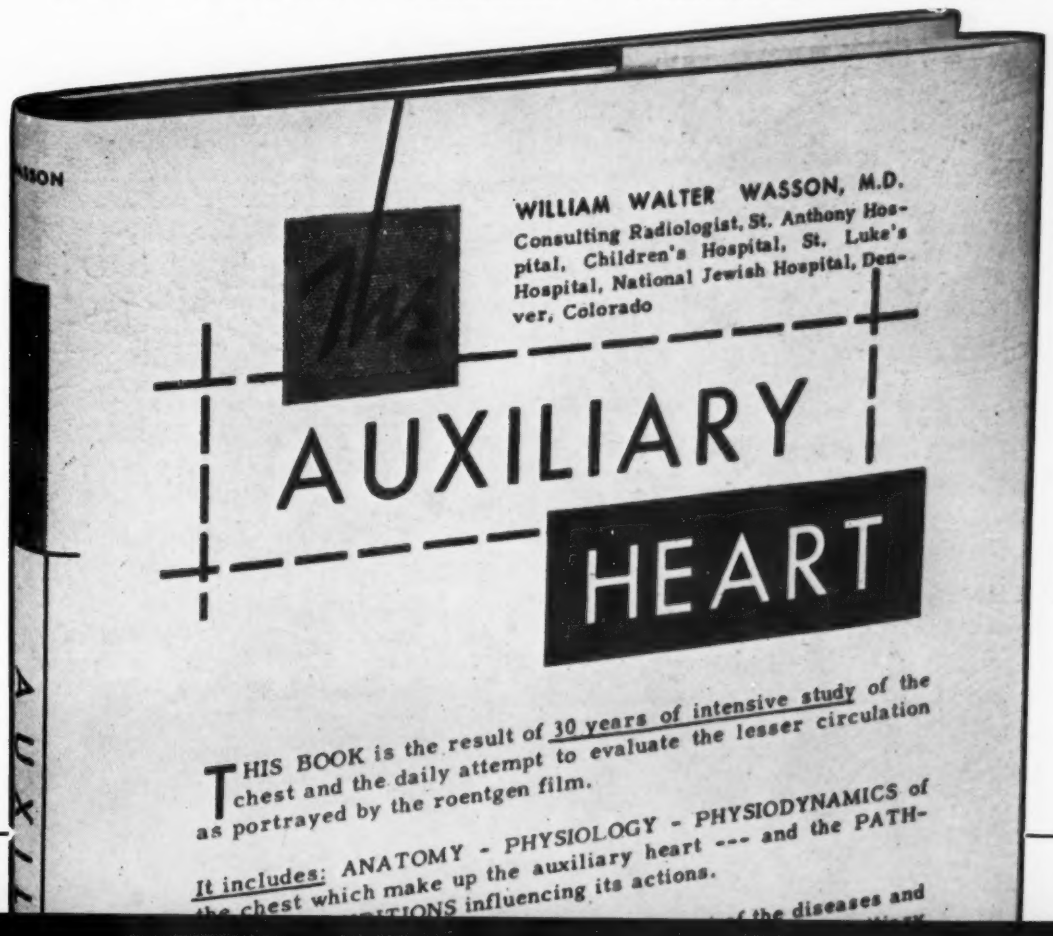
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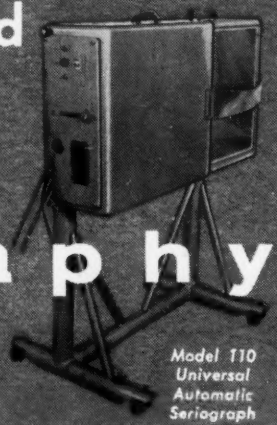

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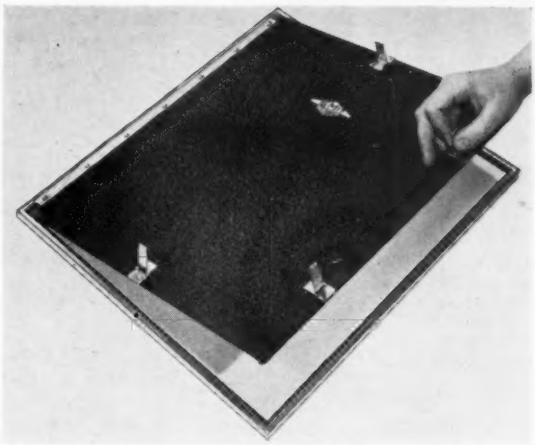

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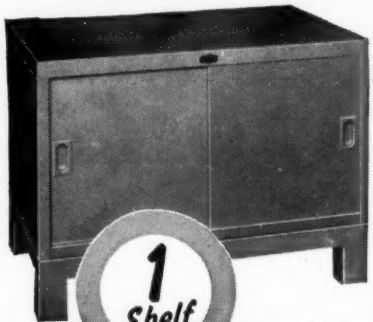

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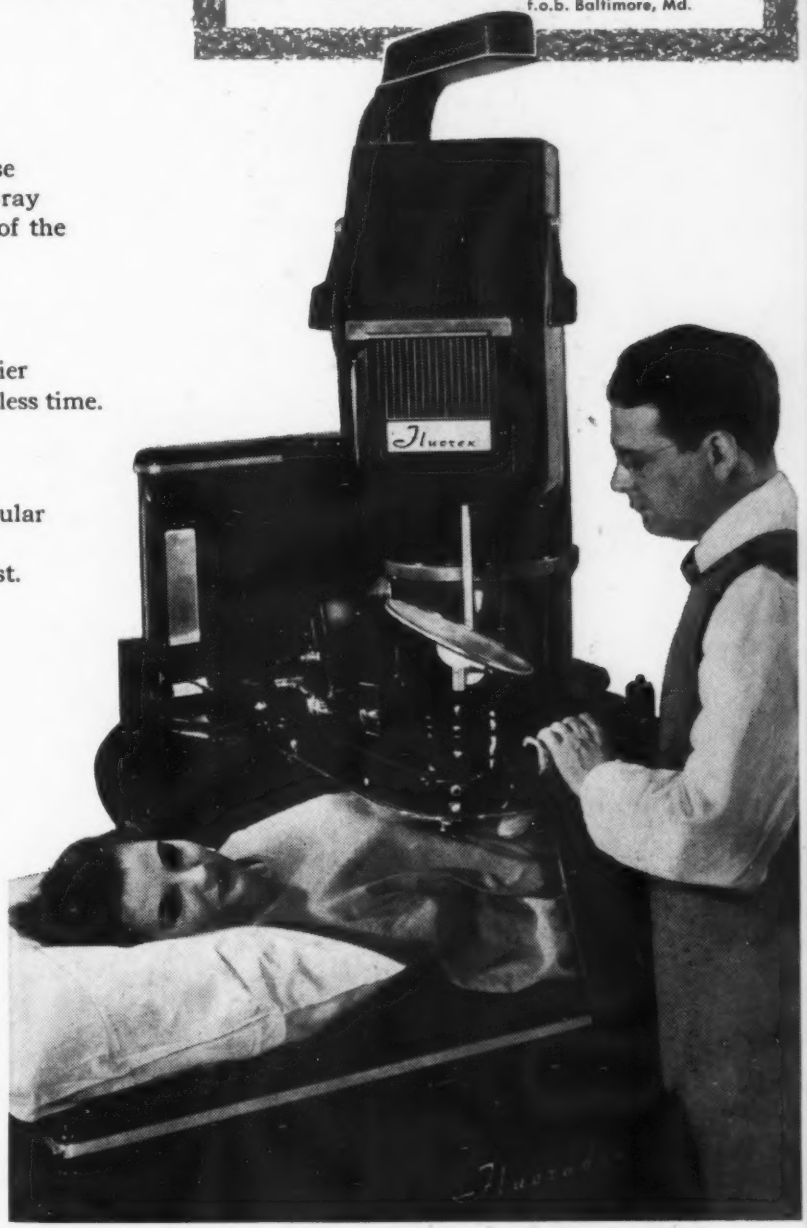

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